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ABSTRACT

These proceedings detail the first of two hearings on the Comprehensive Smoking Prevention Education Act of 1981. This report of the first hearing focuses on health related issues. The report presents testimony from representatives of the administration, from a panel of scientists, and from representatives of the American Cancer Society, American Heart Association, and American Lung Association dealing with the health consequences of smoking and with smoking as an addiction. Testimony from representatives of the Tobacco Institute opposing the legislation is also presented. The proceedings also include a technical review on cigarette smoking as an addiction and an additional 43 statements from health professionals and representatives of the tobacco industry in support of and opposition to the bill. (JAC)

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COMPREHENSIVE SMOKING PREVENTION EDUCATION ACT OF 1981

ED229684

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HEARING

BEFORE THE

COMMITTEE ON LABOR AND HUMAN RESOURCES UNITED STATES SENATE

NINETY-SEVENTH CONGRESS

SECOND SESSION

ON

S. 1929

TO AMEND THE PUBLIC HEALTH SERVICE ACT AND THE FEDERAL CIGARETTE LABELING AND ADVERTISING ACT TO INCREASE THE AVAILABILITY TO THE AMERCAN PUBLIC OF INFORMATION ON THE HEALTH CONSEQUENCES OF SMOKING AND THEREBY IMPROVE INFORMED CHOICE, AND FOR OTHER PURPOSES

MARCH 16, 1982

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COMPREHENSIVE SMOKING PREVENTION EDUCATION ACT OF 1981

TUESDAY, MARCH 16, 1982

U.S. SENATE,
COMMITTEE ON LABOR AND HUMAN RESOURCES,
Washington, D.C.

The committee met, pursuant to notice, at 9:30 a.m., in room 4232, Dirksen Senate Office Building, Senator Orrin Hatch (chairman of the committee) presiding.

Present: Senators Hatch and East.

Also present: Senator Packwood.

OPENING STATEMENT OF SENATOR HATCH

The CHAIRMAN. The committee will be in order.

It is a pleasure for me to welcome you here today to these full committee hearings devoted to the Comprehensive Smoking Prevention Education Act, S. 1929. As chairman of the Labor and Human Resources Committee, I have had the opportunity to learn a great deal about the health needs of our citizens. Over the past decade, there have been numerous Federal initiatives intended to meet these needs, all of which have worthy motives, but with a price tag which has proven to be more costly than the taxpayers can afford.

Consequently, Congress is slowly but surely rediscovering a lost virtue. It is called prudence. And so now, in cooperation with President Reagan, we are trying to target our resources and activities where they are likely to reap the most benefit, where they will give us the "biggest bang for the buck."

In this regard, I have consulted with scientists and health professionals within the public and private sector for their recommendations. I am impressed with their strong consensus favoring more emphasis on preventive medicine and health promotion. We simply cannot afford to spend the lion's share of our Federal health dollar on the treatment of disease, particularly when so many of the illnesses which cause death and disability are preventable.

I speak here not only of the dollar costs but also of the human costs. Our Government's commitment to health prevention must therefore become more than sentimental lip service. Our comment should translate into action. The health of the American people depends on it.

Our Nation's preeminent health experts agree that the most worthwhile single effort we could make to improve the health of our citizens is to convince them of the harmfulness of smoking cigarettes. It causes chronic disease, premature death, and billions of dollars of lost productivity every year. In fact, this legislation enjoys endorsement from virtually every major health and medical professional organization concerned with public health.

I am not interested in legislating health, in telling people how to live their personal lives. I am particularly not interested in having the Federal Government step in if a problem can be solved in the private sector, as it usually can. But I am convinced that the health consequences of smoking are of such magnitude, and that the public is sufficiently unaware of these problems, that we need another major national effort to help our citizens make informed choices about their health. It is not Government regulation, but more public education, I have in mind.

I agree with former Assistant Secretary of Health, Dr. Ted Cooper, when he said, "We will achieve the greatest good not through absolute bans, but by giving our people the knowledge necessary to make rational and informed personal decisions."

That is why Senator Packwood and I have introduced the Comprehensive Smoking Prevention Education Act. Senator Packwood, of course, is with us today, and I am grateful to have him here. He is chairman of the Senate Commerce Committee. We introduced this bill on December 9, 1981.

This bill is designed to stimulate a cooperative effort with the private sector to provide health education on smoking. We would like the Federal Government to continue the activities of the Office of Smoking and Health, which does a commendable job in coordinating and disseminating smoking related information. The stimulus and support that this legislation can provide will further augment these admirable efforts.

In addition, our bill asks the tobacco industry to rotate the messages on the warning labels already mandated by the Federal Cigarette Labeling and Advertising Act. These new labels would inform our smoking population not only of cigarettes' causal link with cancer of the lung, but also with emphysema, a chronic lung disease which causes a slow and agonizing death and disables over 1 million Americans. Other labels will indicate that cigarettes cause heart disease, and may cause miscarriages or premature births. Tobacco manufacturers will also be asked to provide information regarding the chemical additives used as flavor enhancers so that our scientists can determine if these chemicals aren't possibly as cancer-causing as tobacco itself.

[The text of S. 1929 follows:]

II

97th CONGRESS
1st Session

S. 1929

To amend the Public Health Service Act and the Federal Cigarette Labeling and Advertising Act to increase the availability to the American public of information on the health consequences of smoking and thereby improve informed choice, and for other purposes.

IN THE SENATE OF THE UNITED STATES

December 9 (legislative day, November 30), 1981

Mr. Hatch (for himself and Mr. Packwood) introduced the following bill; which was read twice and referred jointly by unanimous consent to the Committees on Commerce, Science, and Transportation and Labor and Human Resources

---

A BILL

To amend the Public Health Service Act and the Federal Cigarette Labeling and Advertising Act to increase the availability to the American public of information on the health consequences of smoking and thereby improve informed choice, and for other purposes.

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled, That this Act may be cited as the "Comprehensive Smoking Prevention Education Act of 1981".

FINDINGS

SEC. 2. The Congress finds that—

(1) cigarette smoking is the largest preventable cause of illness and premature death in the United States, and is associated with the unnecessary deaths of over three hundred thousand Americans annually;

(2) smoking is the primary cause of lung cancer and emphysema in the United States, and is associated with other cancers;

(3) heart disease accounts for nearly one-half of the deaths in the United States, and one-third of the deaths attributable to heart disease are associated with smoking;

(4) the risks of miscarriage, stillbirths, premature births, and child weight deficiencies for pregnant women who smoke are higher than for pregnant women who do not smoke;

(5) certain occupational hazards, in conjunction with smoking, increase substantially the risk of disease and death; and

(6) present Federal, State, and private initiatives have been insufficient in conveying to the American public the information contained in clauses (1) through (5) of this section and other information regarding smoking.

PURPOSE

SEC. 3. It is the purpose of this Act to provide a new strategy to educate and provide information to the American public that will allow individuals to make informed decisions concerning smoking.

SMOKING RESEARCH, EDUCATION, AND INFORMATION

SEC. 4. (a) Title XVII of the Public Health Service Act is amended by adding at the end thereof the following new section:

"SMOKING AND HEALTH

"SEC. 1711. (a) The Secretary shall establish and carry out a program to inform the public of the dangers to health from cigarette smoking. In carrying out such program, the Secretary shall—

"(1) coordinate all activities of the Department which relate to smoking and its effects on health, including research and demonstration projects and educational activities;

"(2) through the Interagency Committee on Smoking and Health established under subsection (b), coordinate the activities referred to in clause (1) of this subsection with all other activities of the Federal Government which relate to smoking and its effects on health;

"(3) through the Interagency Committee on Smoking and Health established under subsection (b), coordinate the activities of the Federal Government referred to in clauses (1) and (2) of this subsection with similar activities in the private sector;

"(4) conduct research and develop new methods for informing the public of the effects of smoking on health, either independently or in conjunction with the private sector, for use in a national effort;

"(5) collect, analyze, and disseminate information, studies, and other data related to smoking and its effects on health;

"(6) make available, through specific publications and bibliographic and reference materials, information on research efforts relating to smoking and its effects on health; and

"(7) undertake any other additional informational and research activities which the Secretary determines necessary and appropriate.

In carrying out the requirements of this subsection, the Secretary shall seek to develop methods of communication with Federal, State, and local entities, as well as with the private sector.

"(b)(1) To carry out the activities described in clauses (2) and (3) of subsection (a), there is established an Interagency Committee on Smoking and Health. The Committee shall be composed of—

"(A) representatives from appropriate institutes and agencies of the Department, which may include the National Heart, Lung, and Blood Institute, the National Cancer Institute, the National Institute on Child Health and Development, the Health Services Administration, the Health Resources Administration, and the Center for Disease Control; and

"(B) at least one representative from the Federal Trade Commission, the Department of Education, the Department of Labor, and any other Federal agency designated by the Secretary.

"(2) The Committee shall meet at least four times each year.

"(c) The Secretary shall transmit a report to Congress not later than January 1 of each year which shall contain—

"(1) current information on the effects of smoking on health;

"(2) an overview and assessment of Federal activities undertaken to inform the public of the effects of smoking on health;

"(3) information regarding the activities of the private sector with respect to the effects of smoking on health; and

"(4) such recommendations for legislation as the Secretary may consider appropriate.".

(b) Section 8 of the Federal Cigarette Labeling and Advertising Act (15 U.S.C. 1337) is amended by striking out subsection (a) and by striking out "(b)" before "The Federal Trade Commission".

CIGARETTE LABELING

SEC. 5. Section 4 of the Federal Cigarette Labeling and Advertising Act (15 U.S.C. 1333) is amended to read as follows:

"LABELING

"SEC. 4. (a)(1) It shall be unlawful for any person to manufacture, import, or package for sale or distribution within the United States, or advertise any cigarettes, the package or advertisement for which fails to bear one of the following statements:

"(A) 'Cigarette Smoking is Dangerous to Your Health. For Information on the Specific Health Consequences of Smoking, Write: Surgeon General, U.S. Public Health Service, Washington, D.C., 20201.'.

"(B) 'WARNING: The Surgeon General has Determined that Cigarette Smoking Causes Emphysema, Lung Cancer, and Other Cancers.'.

"(C) 'WARNING: The Surgeon General has Determined that Cigarette Smoking Causes Heart Disease.'.

"(D) 'WARNING: The Surgeon General has Determined that Cigarette Smoking by Pregnant Women May Result in Miscarriage, Premature Births, or Child Weight Deficiencies.'.

"(E) 'SMOKERS: No Matter How Long You Have Smoked, Quitting Now Greatly Reduces The Risks To Your Health.'.

"(2) The labeling statements specified in paragraph (1) of this subsection shall be rotated on the packages of each brand of cigarettes and the advertisements for each brand of cigarettes in a manner that assures that each of such labeling statements appears an equal number of times on each brand of cigarettes and all such advertisements within the fifteen-month period beginning on the effective date of this subsection and each succeeding fifteen-month period.

"(3) Any labeling statement required under this subsection shall be located in a conspicuous place on every cigarette package and in each advertisement for cigarettes, and shall appear in conspicuous and legible type in contrast by typography, layout, or color with all other printed material on the package or advertisement.

"(4) In accordance with the provisions of section 553 of title 5, United States Code, the Federal Trade Commission shall establish by rule a system to ensure that labeling statements required under this subsection are rotated in accordance with the provisions of paragraph (2) of this subsection, and that at any time each of the five labeling statements appears on at least 15 per centum of all cigarette packages and advertising.

"(b)(1) It shall be unlawful for any person to manufacture, import, or package for sale or distribution within the United States any cigarettes, the package of which fails to disclose the level of—

"(A) tar;

"(B) nicotine; and

"(C) carbon monoxide,

contained in such cigarettes.

"(2) Such tar, nicotine, and carbon monoxide levels shall be based on the results of the most recent tests of cigarettes by the Federal Trade Commission. The Federal Trade Commission shall perform such tests at least on an annual basis.

"(c)(1) It shall be unlawful for any person to manufacture, import, or package for sale or distribution within the United States any cigarettes unless such person has provided to the Federal Trade Commission and the Department of

Health and Human Services a complete list of each chemical additive used in the manufacture of such cigarettes and the quantity of such additive.

"(2) The Federal Trade Commission and the Department of Health and Human Services and any officer or employee thereof shall not disclose to any person outside the Commission or the Department any information received pursuant to paragraph (1).

"(3) For purposes of section 552(b)(4) of title 5, United States Code, and section 1905 of title 18, United States Code, any information received by the Federal Trade Commission and the Department of Health and Human Services pursuant to paragraph (1) shall be considered to be a trade secret.".

PREEMPTION

SEC. 6. Section 5(a) of the Federal Cigarette Labeling and Advertising Act (15 U.S.C. 1334(a)) is amended—

(1) by striking out "statement" each place it appears and inserting in lieu thereof "statements"; and

(2) by inserting before the period the following: "or in any cigarette advertising".

EFFECTIVE DATE

SEC. 7. (a) Except as provided in subsection (b), the provisions of this Act shall take effect on the date of enactment.

(b) The amendments made by sections 5 and 6 of this Act shall take effect upon the expiration of the one-year period beginning on the date of the enactment of this Act. During such one-year period, the Federal Trade Commission shall promulgate such regulations as may be necessary to implement the amendments made by sections 5 and 6 on their effective date.

○

The **Chairman**. Today we are going to hear testimony from representatives of the administration, from a panel of outstanding scientists, and from a distinguished spokesman representing the "big three" volunteer agencies, the American Cancer Society, the American Heart Association, and the American Lung Association.

I would like to take this opportunity to recognize the American Heart Association for their assistance in drafting the Comprehensive Smoking Prevention Education Act.

We will also hear testimony from a representative of the Tobacco Institute as well. The testimony presented today is going to address the health-related aspects of this bill. Senator Packwood has scheduled Commerce Committee hearings for May 10, regarding advertising and trade issues, and I might add, we are very grateful to have Senator Packwood join our committee today.

Senator Packwood and myself, as I have mentioned, have jointly sponsored this legislation, and I certainly appreciate his active interest in these hearings and welcome his participation.

Senator Packwood.

STATEMENT OF HON. BOB PACKWOOD, A U.S. SENATOR FROM THE STATE OF OREGON

Senator **Packwood**. Mr. Chairman, thank you for extending an invitation to me and to all of the members of the Commerce Committee to participate in this hearing on the bill before us. As you are aware, the major purpose of this legislation is to provide consumers with more information about the adverse effects of smoking.

As a member of the Commerce Committee, and now as the chairman of that committee, I have long been concerned with the effects of unfair or deceptive product labeling in advertising, and I am particularly concerned about what I believe is inadequate awareness about the specific health hazards of smoking and about advertising campaigns that target segments of the population such as young adults.

We all know that women who are pregnant and those who take birth control pills may be uniquely affected by smoking and that young adults may be unduly affected by advertising.

The legislation, Mr. Chairman, that you and I have cosponsored has been condemned as an example of burdensome governmental regulation and an abridgment of the first amendment of the U.S. Constitution. This could not be further from the truth. My record on these subjects is clear. I have been a proponent of deregulation in the communication industry, the transportation industry, and where there was a demonstrated need for a freer marketplace, I have supported that deregulation. I have also been a fervent supporter of civil liberties.

But, in the marketplace of information, the American people are desperately in need of more information about the health effects of smoking in order to make a truly free and informed choice.

I look forward to hearing from the witnesses scheduled for this morning and learning more about the health aspects of these issues.

Thank you.

The Chairman. Thank you, Senator Packwood.

I notice that Senator East is with us. Senator, we are happy to invite you to participate in the hearings today.

Senator East. Thank you, Mr. Chairman. I appreciate the opportunity to be with my two distinguished colleagues from the Senate, who are and also distinguished leaders in the Republican Party.

Mr. Chairman, I appreciate, as a member of this committee, the opportunity to make some observations on this legislation. I certainly understand the good intentions of you two distinguished gentlemen, but I will be blunt and categorical about it. I think this is a bad bill.

I would like to briefly state, at the outset, why I think it is a bad bill, and then we can take these matters up with greater precision and focus as we lace our way through the hearings today. I would hope, Mr. Chairman, that in terms of the panels we have here, we can have a balanced presentation this morning, and we shall certainly strive for that end, as I know you will, too, but I would like to say that if it would appear, after the conclusion of our hearings this morning, that additional hearings may be in order so that we can get the broad perspective of opinion on this, I would certainly, as one member of the committee, like to have us move in that direction if it would seem it would be useful as we pursue this subject.

As both of the gentlemen have indicated, the purpose of this bill is to increase the information level of the American people concerning the health hazards of smoking. Now, it is interesting to note that a recent Gallup poll has shown that 92 percent of the American people already know that cigarette smoking does, carried to excess, involve certain health risks, as many things do, such as drinking, excessive use of sugar, salt, fast driving, obesity, and ad infinitum.

My concern here Mr. Chairman, is that you have a bill that is purported to be informational in basic structure and design but, ultimately, if you strip it down to its theoretical base, it has a prohibitional mood about it, because what it would do—and I find this contrary to the philosophy of this administration and to the mandate of 1980—is that this would saddle a legitimate, and honorable, and long-standing industry in this country with an enormous bureaucratic regulation.

We deeply fear that this bill would be used as a frontal assault on an honorable industry by the Government and, also, that it would serve as the basis for very serious assault by the courts upon a very honorable industry.

I would like to put this thing in perspective. There is nothing wrong—in fact, I support, and those who would share my position, I believe, would support the idea—for the Government to inform people about relative health problems, be it tobacco, drinking, salt, sugar, automobiles, obesity, and so forth. This is an appropriate and a useful role of Government.

But again, this bill goes beyond that line. It goes beyond the pale of that legitimate Government responsibility and moves us into an area of excessive, extraordinary involvement by the regulatory side of Government, the executive branch, and also opening up the courts to people who would harass and cause incredible problems, not only for the tobacco industry generally, but for related industries.

Now, there are several points here, Mr. Chairman, and then I shall cease and desist so we can get on with the hearings and get into the particulars of them. I think this bill does great violence to the concept of separation of power.

As chairman of the Separation of Powers Subcommittee of the Judiciary Committee, I think this bill is simply a massive grant of power to the Department of Health and Human Services, frankly, to do about whatever they choose to do.

For example, on page 4 of the act, after listing all of the things that the Secretary of Health and Human Services might do, No. 7 is that all-expansive, open-ended clause that simply is a total invitation to the Federal bureaucracy to move in, lock, stock, and barrel. It says, "Undertake any other additional informational and research activities which the Secretary determines necessary and appropriate."

This is one of the fundamental things taken up in the election of 1980. I think it is contrary to the philosophy of this administration. It certainly is contrary to my philosophy and, I think, to that of the distinguished gentlemen on my right, that we once again, with honorable intentions, mind you—always there are good intentions in this legislation—simply to give this broad, sweeping grant of power to the Federal bureaucracy, which in this case is the Secretary of Health and Human Services.

Eventually, we are going to be back with this problem of having to rein them in. There will be excesses. Again, if we are going to maintain the doctrine of separation of powers, when we give power to the bureaucracy, there ought to be parameters to it; it ought to be carefully spelled out; it ought to be limited and defined. That is a fundamental tenet of separation of power because it is the legislative branch that ought to define the basic policy direction in any area, including this.

Also on this matter of separation of power, I am troubled with the fact that we are told we are going to create another committee. Naturally, of course, I like the title here, "Interagency Committee on Smoking and Health." That is fine, but we are told who will be on this committee, which is a wide range of people, and then, finally, on this matter of separation of power, we are told, at least one representative from the Federal Trade Commission, the Department of Education, the Department of Labor, and any other Federal agencies designated by the Secretary. So the Secretary can do anything he wants to in terms of so-called informational and research activities; he can do anything he wants to in terms of inviting people on board.

I do not want to put it too strongly, Mr. Chairman, but it sounds to me like we are getting up a lynching bee here for the tobacco industry. It is an honorable industry, a legal industry, and a long-term industry in this country. The first tobacco went out of this country from Virginia by John Rolfe back in 1712, so we are not talking about a Johnny-come-lately industry. I will admit I come from a part of the country where this is a part of the heritage, the culture, and tradition. We are a little weary—I will be candid—of being the punching bag for those who simply look upon this as the only health issue in the United States.

Now, on this matter of health findings in this bill, I think what we are going to do, Mr. Chairman, is get into this matter of the subtlety of scientific proof and determination, if you take the very vague way in which it is phrased under section 2. Here we have Congress going on record, proclaiming by edict, very profound scientific conclusions and implications which are going to serve, I fear, as a basis for lawsuits not only against the tobacco industry but to other industries in this country, because it will be charged that Congress has found that if you smoke in addition to working in this kind of industry, it produces this health problem.

So I would remind all industries in this country, not only the tobacco industry, that you open yourself up for serious lawsuits—workmen's compensation, private civil suits, ad infinitum. In other words, it is "welcome everybody aboard," and "anything is fair game."

On this rotational requirement that is provided for here, interestingly, on the House side in the testimony, Mr. Chairman, behavioral scientists pointed out that rotating the advertisements would be counterproductive, because behavioral science has shown, by taking one specific well-worded warning and repeating it is more effective than by confusing and garbling the message. So, curiously, interestingly, the very thing you seek to achieve would be counterproductive.

Again I would get back to this premise: 92 percent of the people, according to the Gallup polls in this country, already know there is a health problem. How much more could you do to inform the American people? That again brings me back to my point of departure. I fear the intention here is not informational, though it is so stated; it is prohibitional, and we are back to, if you will, the Carry Nation mentality as far as smoking goes. Again, the intentions are honorable. I am not questioning that. The intentions are always good in this sort of thing.

But I fear we are going to saddle not only our industry but other industries and the American people with a bill that simply will not work and will do great violence to some fundamental concepts of our Constitution, as well as to what our party, quite candidly, has traditionally stood for.

So again, I appreciate the opportunity, Mr. Chairman, to speak at the length that I have, and I greatly appreciate your indulgence. I might say that Senator Hatch and I, and frequently Senator Packwood and I, agree on many other things, and I greatly respect their judgments and leadership in so many areas. So I would like to make it understood that this is a family feud, and it is not evidence that I am filing divorce proceedings. [Laughter.]

We have not yet gone that far.

The CHAIRMAN. You keep smoking, and we will have a divorce. [Laughter.]

Senator EAST. But they have clearly stepped upon the toes of one of their colleagues, and we expect to respond as responsibly and as effectively as we can at these hearings and any others that are posted, and to make our colleagues know that this is not strictly a "health informational problem." It involves deeper and broader matters than that.

Thank you, Mr. Chairman.

The CHAIRMAN. Well, thank you, Senator East. I know that you feel deeply about this, and we are happy to have you here.

At this time, without objection, we will include the statements of Senators Hawkins and Huddleston in the record.

[Material referred to follows:]

STATEMENT OF SENATOR PAULA HAWKINS
ON
COMPREHENSIVE SMOKING PREVENTION EDUCATION ACT OF 1981

Mr. Chairman, I am pleased that the Labor and Human Resources Committee is holding hearings on S.1929. This legislation, which I have cosponsored, strengthens warning labels on cigarettes, requires disclosure of tar, nicotine and carbon dioxide levels on packages, requires cigarette manufacturers to supply FTC and HHS with a list of the types and quantities of all additives and substances contained in cigarettes and establishes an Interagency committee within the Office of Smoking and Health to coordinate federal activities relating to smoking.

I am delighted with the Administration's decision to support this legislation. Their endorsement is consistent with a desire to reduce illness and death and a desire to reduce the expenditures caused by the adverse health effects of smoking. The Department of Health and Human Services has estimated tht the annual health and economic costs of cigarette smoking will exceed $41 billion in 1981. But the truely sad statistics involve the over 300,000 lives lost each year due to smoking. We must accelerate our campaign to educate the public about smoking, the most preventable cause of death and illness today.

The current warning labels on cigarettes have served a useful purpose in informing the public about the possible adverse health effects of smoking. However, a great deal of additional information and evidence concerning the toxic nature of cigarette smoking has been compiled since the first Surgeon General's report on smoking was issued in 1964. We must continue in our efforts to keep the public informed as to the latest information concerning smoking's effect on lung, larnyx, esophagus, urinary, mouth and pancreatic cancers, emphysema and chronic bronchitis, coronary heart disease, arteriosclerotic peripheral vascular disease, retarded fetal growth and increased risk of prenatal death. We in Congress have a duty to inform the public so they can make informed choices involving their health.

I do not view this bill as imposing regulatory constraints upon the tobacco industry. I veiw it as an educational measure consistent with the Government's duty to warn consumers about adverse health effects of the substances they use.

Statement

of

Senator Walter D. Huddleston
of Kentucky

before the

Committee on Labor and Human Resources
United States Senate

on the

Comprehensive Smoking Prevention Education
Act of 1981

(S. 1929)

*****

Washington, D.C.
March 16, 1982

Comprehensive Smoking Prevention Education Act of 1981

Mr. Chairman, Members of the Committee, I appreciate this early opportunity to present my views on S. 1929, the "Comprehensive Smoking Prevention Education Act of 1981."

I oppose the bill. As introduced this measure is counterproductive and will defeat the principal goals shared by its sponsors. Let me explain why the proponents' good intentions are so misguided in this instance.

Mr. Chairman, cigarette advertising is directed to the smoker -- it's an effort to sell a particular brand to an established consumer. The advertising designed to attract new consumers of proprietary drugs, or dog food, or breakfast cereals, is vastly different in content and style and immediately apparent to the casual observer. As noted in Advertising Age of October 19, 1981, the advertising expenditures of these industries are higher, as a percentage of sales, than those of the tobacco products industry. The "hard sell" is conspicuous by its absence from tobacco products advertising -- and for good reason. The industry is conscious of the controversy about smoking and health, and advertising is styled accordingly.

Over the past ten years, a revolution has occurred in cigarette production and marketing. Low "tar" brands accounted for nearly fifty per cent of all cigarette sales in 1980, compared to two percent just six years earlier. The development of new brands -- low "tar" brands -- and the aggressive advertising of these new brands, has been one industry response to the continuing controversy addressed in these hearings. Another has been the contribution of more than $100 million in unrestricted grants for scientific research on smoking and health.

Who can complain about these voluntary industry initiatives? Who can say the "blunt instrument" approach of government controls and regulation, reflected in S. 1929, will be more productive? I say that S. 1929 will place the progress being made today, under current law and regulation, in the most dire jeopardy.

The premise of S. 1929 is that certain draconian, rotating warning labels against the continued consumption of this lawful product will deter such continued consumption. We simply cannot know whether that assumption is correct or incorrect, but we can cite evidence that it probably is not correct. In Sweden rotating labels have been in effect since 1977 -- and cigarette consumption has increased. In the United States the single warning label has been required since 1966, and its message has become part of the lexicon. And per capita consumption of cigarettes has declined since the single-label requirement was established.

There are several reasons, in my judgment, for the declining per capita consumption of cigarettes in the United States.

First, almost all people, including young people, are informed about the smoking and health controversy. The 1978 Gallup Opinion Index indicated that 90 per cent of the respondents actually believe that "smoking is hazardous to health." The same results were found in the 1978 and 1980 Roper Reports, and in the Chilton Study, as cited by the Federal Trade Commission staff in its May, 1981, report on the cigarette advertising investigation.

Second, the advertising of cigarettes, as I have said, is directed to the brand preferences of current adult smokers.

Third, the young people who begin smoking are responding to peer pressure, not advertising claims, and peer pressure to begin smoking has declined as the smoking and health controversy has been debated over the years.

The rotating labels of S. 1929 may actually spark the rebellious tendencies of American teenagers, as the initial warning label may have done in the 1960's. In testimony before a House committee on March 12th, Dr. Bernice C. Sachs, a noted psychiatrist and President of the Academy of Psychosomatic Medicine, recalled her prediction in 1965 that proposed warnings for cigarette packages and advertising "could well fan the fire of youthful rebelliousness, rather than deter smoking by teenagers." Dr. Sachs continued, "As I predicted, surveys following the adoption of warning statements showed a rise in smoking by children, particularly girls...."

Major educational efforts have not deterred youthful alcohol abuse, teenage promiscuity, or other conduct that is regarded as damaging to young people. These proposed warnings will not stop experimenting with cigarettes either, and an existing program that is relatively balanced should not be cast aside for a new and untried program of potentially counterproductive effect.

Mr. Chairman, I applaud the emphasis on low "tar" cigarettes in brand development and advertising. I applaud the continuing contributions of the industry to unrestricted research on the smoking and health controversy. This committee, with the best of intentions, should not initiate a legislative program which can only impede the voluntary progress that is being made.

There are those who will always smoke cigarettes, no matter what scientific assertions or medical claims are made against the product. And there are those who will be zealots in the war against tobacco, no matter what breakthroughs may be achieved by the manufacturers of cigarettes.

If Congress concludes -- based upon incomplete and inconclusive scientific data -- that the zealots deserve the law's support, the industry's determined efforts to achieve real progress will be demeaned and repudiated in every advertisement they place, on every package of cigarettes they manufacture.

The Congress simply could not have legislated the progress made in the past decade. And Congress should not, at this time, attempt to legislate in a manner that could undermine the basis for the recent, voluntary successes of the industry.

After concrete data are obtained about the true effects of the labeling provisions proposed in this bill, either through in-depth studies or experience abroad, then Congress will be in a position to evaluate rationally the proposition advanced in S. 1929. Until then, the more cautious approach is certainly the most prudent approach.

I respectfully recommend, Mr. Chairman, that S. 1929 be set aside by your committee as an unwarranted and untested intrusion into an industry program of adaptation which has been accepted by the public and has achieved successes not expected by the anti-smoking advocates of a decade ago.

Thank you, Mr. Chairman.

The CHAIRMAN. Our first panel of witnesses represents the administration. And we are very pleased to have them with us. They are Dr. Edward Brandt, the Assistant Secretary for the Department of Health and Human Services; with Dr. Brandt is Dr. C. Everett Koop, the Surgeon General of the United States of America, of the Public Health Service, and Dr. JoAnn Luoto, the Acting Director of the Office of Smoking and Health.

So, Dr. Brandt, why don't we begin with you?

STATEMENT OF EDWARD N. BRANDT, JR., M.D., ASSISTANT SECRETARY FOR HEALTH, PUBLIC HEALTH SERVICE, DEPARTMENT OF HEALTH AND HUMAN SERVICES, ACCOMPANIED BY DR. C. EVERETT KOOP, SURGEON GENERAL, U.S. PUBLIC HEALTH SERVICE AND DR. JoANN LUOTO, ACTING DIRECTOR, OFFICE OF SMOKING AND HEALTH

Dr. BRANDT. Thank you very much, Mr. Chairman, Senator Packwood, Senator East. I am pleased to submit to you today this statement of the Department of Health and Human Services on the health effects of cigarette smoking.

As you pointed out, I am accompanied by Dr. C. Everett Koop, the Surgeon General of the Public Health Service, and Dr. JoAnn Luoto.

Let me begin by presenting a capsule description of the health effects of cigarette smoking and then a somewhat more detailed description of smoking and its relationship to cancer and cardiopulmonary diseases.

In summary, cigarette smoking is clearly the single most important preventable cause of premature illness and death in the United States. Estimates of the number of deaths related to smoking exceed 300,000 annually. One may compare this figure with the 105,000 deaths that occur each year as a result of injuries, 20,000 deaths from homicides, or the 40,000 infant deaths.

Cigarette smoking is one of the three major independent risk factors for coronary heart disease and for arteriosclerotic peripheral vascular disease. It is a major cause of cancer of the lung, larynx, oral cavity, and esophagus and a major cause of chronic bronchitis and emphysema.

Maternal cigarette smoking is associated with retarded fetal growth, an increased risk for spontaneous abortion and for prenatal death, and slight impairment of growth and development during early childhood.

Cigarette smoking acts synergistically with oral contraceptives to enhance the probability of coronary and some cerebrovascular disease; with alcohol to increase the risk of cancer of the larynx, oral cavity, and esophagus; with asbestos and some other occupationally encountered substances to increase the likelihood of cancer of the lung; and with other risk factors to enhance cardiovascular risk.

Mr. Chairman, cancer was the first disease to be associated with cigarette smoking. As Dr. Koop pointed out in introducing our 1982 report on smoking and health a few weeks ago, reports linking smoking and lung cancer began appearing in the scientific literature as long as 50 years ago. In 1964, when the Surgeon General's Advisory Committee's report was issued, lung cancer in men and chronic bronchitis in both men and women were the two diseases which the committee identified as being caused by cigarette smoking.

The evidence which links cigarette smoking with lung and other cancers was reviewed in the most careful detail in the 1982 report just issued. Today, 18 years after the 1964 report, additional human experience and enormous amounts of new research make it possible for science to conclude that cigarette smoking is a major cause of cancers of the lung, larynx, oral cavity, and esophagus, and that it is a contributing factor in the development of cancers of the bladder, pancreas, and kidney.

Lung cancer accounts for one out of every four cancer deaths, and 85 percent of these are due to smoking. Overall, approximately 30 percent of all cancer deaths are attributable to cigarette smoking.

A subject which was hardly touched upon in the 1964 report is the effect of smoking on women and, in the case of maternal smoking, its effect on the fetus and infant. In 1980, this was the topic of the Department's report to Congress. Its conclusions were that women are not immune to the damaging effects of smoking and that the lesser occurrence of smoking-related diseases among women smokers is a result of their having lagged about 25 years behind men in their widespread use of cigarettes.

The 1980 report further established that cigarette smoking is a major threat to the outcome of pregnancy and the well-being of the baby.

Another public health question, now enormously important, relates to the use of the new, low-yield cigarettes. This was the subject of the Department's 1981 report. The conclusions were that although there is no safe cigarette, smoking cigarettes with lower yields of tar and nicotine poses a lower risk of lung cancer than smoking higher yield cigarettes, provided there is no compensatory change in smoking patterns.

The 1982 report noted that the more than 100 diseases we call cancer are the second leading cause of death in the United States. It further concluded that smoking is causally related to at least 30 percent of all cancer deaths and that these relationships are quite strong.

Let me mention one program of the Office of Cancer Communications, because we are very proud of its success. Based on the well-documented evidence that counseling by a physician can motivate smokers to quit, the office developed a kit for physicians to use with patients. More than 135,000 of these kits have been distributed. The kit was so well received that a similar one has now been created for dentists.

Chronic obstructive pulmonary disease today represents the fastest growing of the major causes of death, now ranking fifth. In 1980, 55,000 Americans died of pulmonary disease. Almost 3 million Americans now suffer from emphysema, a terribly debilitating disease. The evidence is substantial and unequivocal that cigarette smoking is the chief culprit in the onset or exacerbation of emphysema and chronic bronchitis.

During the past 10 years, we have obtained a far better understanding of the mechanisms of lung damage, including the destruction of elastin, a major structural protein of the lung which is adversely affected by cigarette smoke.

Despite a dramatic decline in mortality during the past decade, coronary heart disease remains the No. 1 killer in this country, claiming three-quarters of a million lives in the United States each year. Cigarette smoking is one of the three major risk factors for coronary heart disease, the other two being high blood pressure and high serum cholesterol.

Further evidence indicates that the more one smokes, the greater the risk. The exact mechanisms of how cigarette smoking affects coronary heart disease are still unknown and are the subject of considerable research now underway. Nevertheless, the evidence based on epidemiologic and autopsy studies clearly linking the amount of smoking with higher incidence of heart disease is indeed impressive.

Mr. Chairman, Dr. William Pollin, Director of Drug Abuse, will appear on the next panel to address the serious issues of the addictive nature of smoking.

Let me now ask Dr. Koop to give a brief presentation on the 1982 Surgeon General's report, following which we will be happy to respond to questions.

The CHAIRMAN. Dr. Koop, we are happy to have you with us.

Dr. KOOP. Mr. Chairman, Senator Packwood, Senator East, for the sake of brevity, I will try to condense this rather lengthy report into a discussion of the two charts on your right, and we will just be talking about death and about cancer.

The first chart over there confirms what Dr. Brandt has just said, that cancer of the lung, larynx, oral cavity, and esophagus are those that have been known to be linked with smoking now for many years, and these are two old fashioned comparative pie diagrams which compare 1950 with 1977. In each instance, the blue pie is all cancer deaths, and in each instance the red slice is the percentage of those cancer deaths which are due to lung, larynx, oral cavity, and esophagus, and you can see very graphically there the increase in proportion of the deaths from cancer due to those four.

Please remember that we have additional evidence that smoking is linked to other types of cancer, but they are not included in any way on this diagram.

If you would then turn your attention to the chart that is closer to you, this shows time trends in cancer mortality. Simply look at those three lines. The top red line shows the mortality for cancer of all types of cancer, and you can see that as far as this graph is concerned, from 1950 to 1978, there has been a steady swing upward of mortality from all cancers.

Below that, you will see a blue line that deals with lung cancer only, and that line is ever so much more steep than is the other one. Then the bottom red line shows all cancers, other than lung, have been decreasing from 1950 to 1978 and, indeed, were it not for that blue intervening line of lung cancer, then the upper line of all cancer would also have been coming down with a slope approaching that of the other cancers.

The reason that the lower red line shows that rather steep slope downward is due to the fact that we have made progress against other cancers such as prostate, rectum, colon, and breast.

And, finally, I would call your attention to the fact that if you look at the last 3 years of that slide, the slope of all cancer parallels exactly the slope of lung cancer at the same time that other cancers remain absolutely flat. So I think the conclusion is self-evident there: smoking and cancer are related in that very specific way.

I would like to provide for you, Mr. Chairman, a copy of the Surgeon General's report for the record if you so desire.

The **Chairman**. Thank you. I appreciate that.

[The prepared statement of Dr. Brandt follows:]

DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

Office of the Assistant Secretary
for Health
Washington DC 20201

FOR RELEASE ONLY UPON DELIVERY

STATEMENT BY

EDWARD N. BRANDT, JR., M.D.
ASSISTANT SECRETARY FOR HEALTH
PUBLIC HEALTH SERVICE
DEPARTMENT OF HEALTH AND HUMAN SERVICES

BEFORE THE

SENATE LABOR AND HUMAN RESOURCES COMMITTEE

MARCH 16, 1982

Mr. Chairman and Members of the Committee:

I am pleased to submit to you today this statement of the Department of Health and Human Services on the health effects of cigarette smoking. These health effects and their significance to the American people must necessarily provide the rationale and justification for whatever action your Committee may take in regard to the bill before you.

With me today are Dr. C. Everett Koop, Surgeon General, and Dr. Joanne Luoto, Acting Director, Office on Smoking and Health.

I will begin by presenting a capsule description of the health effects of cigarette smoking and then a more detailed description of smoking and cancer and cardiopulmonary diseases. I will also address research efforts by the National Institute on Drug Abuse on the addictive characteristics of cigarette smoking.

In summary, cigarette smoking is clearly the single most important preventable cause of premature illness and death in the United States. Estimates of the number of deaths related to smoking exceed 300,000 annually. One may compare this figure with the 105,000 deaths that occur each year as a result of all injuries, 20,000 deaths from homicides, or the 40,000 infant deaths.

Cigarette smoking is one of the three major independent risk factors for coronary heart disease and arteriosclerotic peripheral vascular disease; a major cause of cancer of the lung, larynx, oral cavity and esophagus; and a major cause of chronic bronchitis and emphysema.

Cigarette smoking is a contributory factor in cancer of the urinary bladder, kidney, and pancreas. It is also associated with peptic ulcer disease. Maternal cigarette smoking is associated with retarded fetal growth, an increased risk for spontaneous abortion and prenatal death, and slight impairment of growth and development during early childhood.

Cigarette smoking acts synergistically with oral contraceptives to enhance the probability of coronary and some cerebrovascular disease; with alcohol to increase the risk of cancer of the larynx, oral cavity, and esophagus; with asbestos and some other occupationally encountered substances to increase the likelihood of cancer of the lung; and with other risk factors to enhance cardiovascular risk.

Involuntary or passive inhalation of cigarette smoke can precipitate or exacerbate symptoms of existing disease states, such as asthma and cardiovascular and respiratory diseases and may be carcinogenic for nonsmokers. Smoking is also the major identifiable cause of deaths and injuries from residential fires.

Mr. Chairman, cancer was the first disease to be associated with cigarette smoking. As Dr. Koop pointed out in introducing our 1982 report on smoking and health a few weeks ago, reports linking smoking and lung cancer began appearing in the scientific literature as long as 50 years ago. In 1964, when the Surgeon General's Advisory Committee's report was issued, lung cancer in men, and chronic bronchitis in both men and women, were the two diseases which the Committee identified as being caused by cigarette smoking.

The evidence which links cigarette smoking with lung and other cancers was reviewed in the most careful detail in the 1982 report just issued. Today, 18 years after the 1964 report, additional human experience and enormous amounts of new research make it possible for science to conclude that cigarette smoking is a major cause of cancers of the lung, larynx, oral cavity, and esophagus, and that it is a contributory factor in the development of cancers of the bladder, pancreas, and kidney. Lung cancer accounts for one out of every four cancer deaths, and 85 percent of these are due to smoking. Overall, approximately 30 percent of all cancer deaths are attributable to tobacco use.

A subject which was hardly touched upon in the 1964 report is the effect of smoking on women, and in the case of maternal smoking, its effect on the fetus and infant. In 1980, this was the topic of the Department's report to Congress. Its conclusions were that women are not immune to the damaging effects of smoking, and that the lesser occurrence of smoking-related diseases among women smokers is a result of women having lagged one-quarter century behind men in their widespread use of cigarettes.

The 1980 report established that cigarette smoking is a major threat to the outcome of pregnancy and the well-being of the baby. The risk of spontaneous abortion, fetal death, and neonatal death increases directly with increasing levels of maternal smoking during pregnancy. Smoking causes a markedly increased risk of heart attack and subarachnoid hemorrhage.

Another public health question, now enormously important, relates to the use of the new, low-yield cigarettes. This was the subject of the Department's 1981 report. The Report's conclusions were that although there is no safe cigarette, smoking cigarettes with lower yields of tar and nicotine poses a lower risk of lung cancer than smoking higher-yield cigarettes, provided there is no compensatory change in smoking patterns. Increasingly, smokers have turned to these lower-yield products; there is evidence to suggest that in doing so, at least some have increased their smoking or changed the way they smoke. This may have negated any potential benefit in their having switched to these products.

Smoking and Cancer

The 1982 Report of the Surgeon General on the Health Consequences of Smoking focussed upon cancer. The report noted that the more than 100 diseases we call cancer are the second leading cause of death in the United Sates. The report also made these important points:

- It is now clear from a large number of epidemiologic studies--both retrospective and prospective--that smoking is causally related to at least 30 percent of all cancer deaths. This means that approximately 129,000 people a year die of cancers related to smoking.

- The causal relationships are strong. If we just look at lung cancer, the major cause of cancer death among U.S. males, a cigarette smoker is 10 times more likely to die of this disease than a nonsmoker. And this risk increases with the number of cigarettes smoked--a direct dose-response relationship.

- For the first time, two preliminary epidemiologic studies are suggesting an increased risk of lung cancer in nonsmoking wives of smoking husbands, implicating sidestream smoke as a cancer risk factor.

- We are encouraged by the figures on people who have been able to quit smoking, most of them through their own efforts. In 1965, 42 percent of adults in the United States smoked. In 1980, the proportion dropped to 33 percent.

I would like to mention one program of the Office of Cancer Communications because we are proud of its success. Based on the well-documented evidence that counseling by a physician can motivate smokers to quit, the office developed a "Helping Smokers Quit Kit" for physicians to use with patients. More than 135,000 of these kits—which include posters for the waiting room, take-home materials for smokers, and information on counseling the smoking patient—were distributed. In fact, the kit was so well received that a similar one was created for dentists. This project was endorsed by the American Dental Association, which is cooperating with us to distribute the kit. Staff are now working with the American Pharmaceutical Association to develop a similar program for pharmacists.

The National Cancer Institute is interested in pursuing some new leads. There is a growing body of evidence that people who smoke low tar and nicotine cigarettes adjust their smoking behavior--inhaling more deeply or covering the ventilation holes in the cigarette filters. This is thought to be an attempt to compensate for the decreased nicotine yield. We plan to take a look at this question. If these preliminary studies are confirmed, it would imply that smokers of todays' cigarettes are not decreasing their exposure to nicotine and in fact may actually be increasing their exposure to harmful combustion products such as hydocarbons and carbon monoxide.

Smoking and Cardio-pulmonary Disease

Chronic obstructive pulmonary diseases (COPD) today represents the fastest growing of the major causes of death, now ranking fifth. In 1980, 55,000 Americans died of pulmonary diseases. Almost three million Americans now suffer from emphysema, a terribly debilitating disease. More than seven million have chronic bronchitis. Chronic respiratory diseases account for approximately ten percent of disability benefits for lost work hours. And, the evidence is substantial and unequivocal that cigarette smoking is the chief culprit in the onset or exacerbation of these diseases.

Research has for some time provided us with data demonstrating that smokers have higher mortality rates from chronic bronchitis and emphysema and that smokers have far less pulmonary function than nonsmokers. During the past ten years, we have also obtained a far better understanding of the mechanisms of lung damage, including the destruction of elastin, a major structural protein of the lung which is adversely affected by cigarette smoking. And, within recent years, evidence has been reported which suggests that the small airways function of the lung may be adversely affected in healthy nonsmokers if they are exposed to cigarette smoke from others.

Research continues in this area to give us a better basis of knowledge in order to prevent or arrest the progress of pulmonary diseases. Studies have demonstrated the benefits of smoking cessation, including improvements in lung performance on standard spirometric (breathing) tests soon after one quits. However, pulmonary diseases represent a progressive condition and once a certain point is reached we can only hope to retard its progression. Investigators are working towards developing a simplified means of detecting the disease condition at an early enough stage to intervene and reverse the process. At the same time, research continues to try to develop and evaluate programs designed to help individuals give up smoking, since smoking prevention or cessation represents the only effective intervention measure we now have.

Despite a dramatic decline in mortality during the past decade, coronary heart disease remains the number one killer in this country, claiming three-quarters of a million lives in the United States each year. For every minute of the day, there are about three Americans who suffer a heart attack. While the progress in reducing coronary heart disease and other cardiovascular deaths during recent years is heartening, the scope of the problem remains enormous.

Cigarette smoking is one of the three major risk factors for coronary heart disease; the other two being high blood pressure and high serum cholesterol. Epidemiological evidence clearly places the smoker at a higher risk of heart disease than the nonsmoker. The more one smokes, the greater the risk. There is also evidence that smoking cessation can decrease the risk. After only one year free of cigarettes, a former smoker may be able to reduce the risk of heart disease to close within that of the nonsmoker.

The exact mechanisms of how cigarette smoking affects coronary heart disease are still unknown and are the subject of considerable research now underway. Nevertheless, the evidence based on epidemiologic and autopsy studies clearly linking the amount of smoking with higher incidence of heart disease, is indeed impressive.

The **Chairman**. Dr. Brandt, we appreciate your very informative testimony.

How likely is it that the average person in this country would understand all of the health risks involved with smoking cigarettes from present warnings?

Dr. **Brandt**. I think that the major association or major thing that most people associate with cigarette smoking is lung cancer. Certainly the American Heart Association has expressed concern, that more people are not aware of this being one of the three major risk factors in heart disease, and I think only those families, probably, that have connection with someone who has emphysema are aware of that risk.

So I think people are aware that there are adverse health effects associated with cigarette smoking, but I do not think they are as aware of the specific situations.

The **Chairman**. The data you presented regarding the health consequences of smoking, I think, would be frightening to anybody. You have said that the causal relationship is strong. Could you explain again how it is determined that cigarette smoking causes the diseases that you identified?

Dr. **Brandt**. The vast bulk of the evidence is epidemiologic in nature. Epidemiology is that science which is oriented to attempting to find the causal factors and contributing factors to the development of disease. Clearly, the evidence from such studies must be evaluated and must be examined on the basis, really, of five criteria, one being the consistency of these findings; that is, in repeated studies of various populations and various groups, you continue to find a higher incidence of the disease among smokers than nonsmokers.

Second, the strength of that association. One needs to be sure that the association is in fact strong and that it can be consistently identified.

Specificity of the association; the temporal relationship. Clearly, if cigarette smoking results in disease, then the disease must follow the cigarette smoking.

And, finally, the coherence of the association; namely that there should be some biological explanation for these things, including what is most commonly used, the dose-response relationship, and the dose-response relationship of cigarette smoking with those illnesses is quite well demonstrated. That is, the more you smoke, the greater the likelihood of the disease.

The **Chairman**. Obviously, a great deal of research has been done relating to smoking and health, and the great majority of scientific evidence does conclude that smoking is indeed harmful to health.

Nonetheless, it seems to me some scientists do not accept the conclusion that cigarette smoking is the cause of disease and death, so how do you account for their skepticism?

Dr. **Brandt**. I think some scientists clearly look for complete identification of the mechanism before they will accept factors as being causal. As I pointed out in my testimony, at the present time, the exact physiologic or pathophysiologic mechanisms for cigarette smoking in these illnesses has not been clearly demonstrated, but certainly medicine, throughout history, has identified problem areas and other areas to intervene to in fact prevent the development of disease, and I think the evidence is just irrefutable that if you get people to stop smoking, the risk of their developing these diseases goes down dramatically, so that it seems clear that the action that is warranted is in fact to encourage people to quit smoking or not to begin because their risk is certainly reduced.

The **Chairman**. Thank you.

Senator Packwood.

Senator **Packwood**. Dr. Brandt, I was struck by the similarity of your testimony 5 days ago before Congressman Waxman's committee and your testimony here today. Indeed, as I go through the testimony there and your testimony here, it is clear that it was typed on the same automatic typewriter. Page 1 is identical; page 2 is identical. You expand your testimony a bit in the House, but you also have in the House testimony:

> We support the bill's requirements for strong health warnings because we believe they would increase the public knowledge of the hazards of smoking and make it possible for smokers and potential smokers to make better informed judgment as to whether to continue or begin smoking.

But the typewriter seems to have run out of ribbon before you finished your testimony here. Is that still your position?

Dr. **Brandt**. The position is that we certainly support stronger health warnings about cigarette smoking, yes, sir.

Senator **Packwood**. Well, now, in the testimony in the House, you said, "We support the bill's requirements for strong health warnings." Is that still your position?

Dr. **Brandt**. Well, I think that what we are concerned with, as I tried to expand in some of the questions and answers, is the concept of having stronger health warnings. I think, in that testimony, you will find that we disagreed with some of the specifics of that bill, and in fact the reason that the testimony today is different is because we addressed it strictly to the health consequences and not to any specific piece of legislation.

Senator **Packwood**. Well, now, let me ask you this. Is there any change in the degree of your support for this bill as opposed to the House bill, which is indeed a slightly stronger bill than this?

Dr. **Brandt**. I am sorry?

Senator **Packwood**. The House bill has two provisions in it that this bill does not have. One is allowing private suits to enforce the remedy on labeling disclosure, and the other is public disclosure of the cigarette contents, whereas our bill does not have the private suits and it has private disclosure to the Federal Trade Commission.

But, most specifically, I am curious if you still support the bill's requirements for the strong health warnings.

Dr. **Brandt**. We support having stronger health warnings, yes, sir.

Senator **Packwood**. Is there any difference between your position last week and this week on the health warnings?

Dr. **Brandt**. I am sorry, on the health warnings?

Senator **Packwood**. On the health warnings on the packages of cigarettes.

Dr. **Brandt**. I don't think there is any difference in that we certainly support stronger health warnings and, furthermore, a total program of public education, which the Public Health Service is attempting to carry out.

Senator **Packwood**. Let's go down our bill, then. Can you tell us which parts of our bill the administration endorses and which parts they want to change?

Dr. **Brandt**. I cannot tell you which parts in specific, Senator Packwood, because in fact the bill report for this bill has not been completed, to my knowledge. Therefore, I can only support the concept of stronger health warnings.

Senator **Packwood**. Say that again. The bill report is what?

Dr. **Brandt**. To my knowledge, the bill report has not been completed; at least I have not been provided with such a report.

Senator **Packwood**. The bill report. Do you mean by your department?

Dr. **Brandt**. The analysis of it, yes, sir.

Senator **Packwood**. Well, all right. You did support, 5 days ago, the House bill's requirements for the strong health warnings.

Dr. **Brandt**. Well, we supported that bill with the reservations that are included in the testimony and in the questions.

Senator **Packwood**. All right. Let's go through those reservations. Do you support the mandatory requirement of the placing of the warnings on the cigarette package?

Dr. **Brandt**. If that is the most effective way to accomplish it, yes.

Senator **Packwood**. Do you support that?

Dr. **Brandt**. I personally support the idea that we must have stronger health warnings.

Senator **Packwood**. Does the administration support that?

Dr. **Brandt**. As I say, the specifics of this bill—I mean the bill that you are talking about—certainly support the concept of stronger health warnings. Yes, sir.

Senator **Packwood**. Doctor, then tell me what your statement means before Congressman Waxman's committee. "We support the bill's requirements—the bill's requirements—for strong health warnings, because we believe," and so on. What does that mean?

Dr. **Brandt**. It means——

Senator **Packwood**. Do you know what is in his bill?

Dr. **Brandt**. I am sorry?

Senator **Packwood**. Do you know what is in the House bill?

Dr. **Brandt**. Yes, I do.

Senator **Packwood**. And do you know how strong the health warnings are in that bill?

Dr. **Brandt**. Yes, sir.

Senator **Packwood**. And you know that the ones in our bill are slightly weakened from that, not the specific warnings but the bill is slightly weaker than the House bill.

Dr. **Brandt**. You will note also, sir, that in that testimony, we requested or we disagreed with the specific wording of those warnings and in fact suggested that if warnings are to be placed on cigarette packages, that the Secretary of HHS be permitted to design the specific wording.

Senator **Packwood**. Now, where does it say that?

Dr. **Brandt**. I don't have a copy of the testimony.

Senator **Packwood**. All I have in your testimony from last week is that you were opposed to the creation of a separate Office of Smoking and Health; you thought that was redundant and unnecessary. You indicate that the Secretary be given the authority for possibly modifying the actual wording.

Dr. **Brandt**. Yes.

Senator **Packwood**. Again, I do not think we would quarrel with that so long as there is a mandatory requirement for the warning. I am not sure that what Senator Hatch and I would draft would necessarily be the health warnings you might want on the package 2 or 3 or 4 years from now, as you have had more research coming in. There may be other things we find, and this does not have to be written in stone.

I am not trying to unduly badger you, Doctor, but I want to make sure that there has been no change in the administration's position between last week and this week, because we were under the impression that the administration supported the health warnings, varying the wording slightly, but supported them, and I sense some difference now from last week.

Dr. **Brandt**. The administration's position is that it supports stronger health warnings. The specific wording of those warnings, and so forth, and the way in which they are to be used is something that is still being studied.

Senator **Packwood**. Now, the way in which they are to be used. Do you mean whether or not they will be compulsorily required to be put on cigarette packages is still an issue being weighed?

Dr. **Brandt**. Yes, it is.

Senator **Packwood**. Have you talked with anybody between last week and this week about your testimony, anybody in the administration?

Dr. **Brandt**. Have I?

Senator **Packwood**. Yes.

Dr. **Brandt**. Well, I have talked to various members of my staff as well as the Secretary.

Senator **Packwood**. Have you talked with anybody who suggested that the statement that you made last week was too strong, and perhaps it should be toned down?

Dr. **Brandt**. Nobody has spoken directly to me about that, no.

Senator **Packwood**. Has anybody spoken indirectly to you about that? [Laughter.]

Dr. **Brandt**. The testimony that I gave today has been completely approved by the administration.

Senator **Packwood**. It is substantially weaker testimony than last week. You have left off the whole part of the testimony about, "We support the bill's requirements for stronger health warnings," out of your testimony this week.

Dr. **Brandt**. On the other hand, Senator Packwood, we have come here principally to discuss not the legislation but the health consequences of smoking.

Senator **Packwood**. Let me ask your personal opinion, not your administration position. Do you personally think these health warnings are necessary?

Dr. BRANDT. My personal opinion is that the health warnings to prevent cigarette smoking and to encourage people to stop are effective in accomplishing that goal, yes.

Senator PACKWOOD. And you think they are effective on the cigarette packages and on the advertising?

Dr. BRANDT. I think that the evidence that we have now, in my personal opinion, is that it has been effective.

Senator PACKWOOD. Thank you, sir. No other questions.

Senator East.

Senator EAST. Ladies and gentlemen, I wish to, as a member of the Labor Committee, welcome you here this morning, and we appreciate your taking the time to come and discuss with us what I think is a very important subject, not only in terms of the area of the country that I come from but, of course, in terms of an item of considerable national interest and concern.

I think that the two of you together somewhat symbolize the difficulty of the problem we face; that is, Dr. Koop coming at this problem from the standpoint of scientific causation, which I would like to pursue briefly with him, and then I think Dr. Brandt symbolizes, of course, in his position, the question of proper responsibility for government in terms of scientific evidence that it has; what it ought to do with it? Should it inform, should it go further into other things, and so forth?

Let me start, if I might, with this question, Dr. Koop, of scientific causation. My background has been in law and the behavioral sciences, social sciences, as opposed to your field of medical science, but there are certain common threads that I think are of our interest and that run through this.

Let me just state my position briefly, and I would appreciate your reaction to it. We all know that problems of causation and the scientific establishment of causation are, again depending upon the subject matter, but quite frequently can be, intricate, difficult, complex. Solutions do not yield themselves up easily. "Intractable" is a word we might use to identify this problem of identifying scientific causation in so many areas.

A word that I know is used a lot in your field of physical sciences, natural sciences, medical science, and of course we use it in the social sciences and behavioral sciences, is this idea of variables, that in any given context in which you are trying to establish scientific causation, what greatly ups the probability or improbability of determining and establishing causation is this problem of variables, and particularly the independent variables, the ones that you cannot isolate.

We have a terribly difficult time with that in the social sciences. In politics, for example, behavioral studies in politics, you have an enormous problem of isolating all the variables because you cannot hold them constant, and so we try, at least in political science, in the behavioral science there. to appreciate the enormous limitations that we have frequently to establish the scientific causation.

A problem I have with this, and with the bill in particular, but just generally, on this question of scientific causation, is that, for example, in the findings that we are asked to conclude here, we are told that Congress finds that—somewhat like a laboratory report—cigarette smoking is the largest preventable cause of illness and premature death; smoking is the primary cause of lung cancer and emphysema, heart attacks, miscarriage, stillbirths, premature births.

Then we are told, for example, in number 5, certain occupational hazards in conjunction with smoking increase substantially the risk of disease and death. Then we are told present Federal, State, and private initiatives have been insufficient in conveying to the American people the information contained in clauses 1 through 5, and so forth.

I realize we cannot unravel all of this, but as I look at this list of so-called scientific findings, it brings me back to my training in the social sciences and behavioral sciences, where we would just be very, very reluctant to make such broad, grandiose conclusions. In short, it strikes me that maybe what we are doing here is stating conclusions in terms of suppositions, but we could not say, in a genuine scientific sense of isolating the variables, weighing them, that we have truly scientifically established all of these things.

My point is that where science allows itself to be politicized, and I think it is here, quite candidly—I don't mean you are but I mean the potential of this bill—where you politicize science, in the long run, I think you do a great disservice to science and, more particularly, the public begins to be suspicious of so-called scientific findings.

For example, several years ago, we were told that nitrites in bacons, meats, and so forth, were cancerous. Cut out bacon, cut out meats, and so on and so forth. The other day I read a report, and I heard it discussed in great depth by a scientist on a radio talk show, in which he said this is no longer the case, that it has now been determined subsequently, through more careful examination, that the small amount of nitrites found in terms of foods and so forth are so infinitesimal, the health risk is negligible, nonexistent.

But on that point, several years ago, the American people were just panicking on this question of eating bacon and canned meats and bologna and that sort of thing. This occurred more recently on saccharin. Now the warning label is on all of your diet colas, that this may be hazardous to your health, or something like that.

Again, I read a report the other day and understand that now we are less sure that saccharin really does present a serious health problem in the United States. So again, I query whether we didn't overreact there.

Now, here again, I want to make clear to you and to everyone else that I personally favor the approach of taking genuine, established scientific fact, which I think your area can do, as Surgeon General, and informing the American people about these findings in an objective, civil, low-keyed way. I think this will be more effective in terms of improving health quality in this country than by being pulled into groups and movements that I think go beyond the pale. I think they begin to read their own wishes and values and conclusions into alleged scientific finding.

It is trendy today to be antitobacco, antismoking. I understand that, just as some years ago it was trendy to be antithis or antithat. I don't know what it will be next. Time magazine has a big article on salt now, not the Strategic Arms Limitations salt but the white stuff. It is bad for you, and maybe we will have to have more

Government information on that. I certainly hope we would not have our salt shakers taken away from us. The same goes for our sugar bowls. We will want more information on that.

I think the scientific findings of the Surgeon General, as turned over to Dr. Brandt and his department, and the great work that Secretary Schweiker is doing to inform us of those things, combine to perform a valuable public service. It is a proper bringing together of science and Government. But again, when we get into this prohibitional mentality, and everybody is sucked into the maelstrom, including science, and asked to conclude things that, if you really got them down and sat them down and got them in the corner and said, gentlemen, as you understood science in medical school, as you understand the very difficult and intractable world of establishing scientific correlation, we are uncomfortable with it. We may in fact have prostituted ourselves a bit in order to be a part of this so-called great movement that has great and good and honorable intentions.

In the case of Dr. Brandt—and then I will let the two of you respond to it—in terms of the responsibility of your department—and I appreciate there is one, and I might note that I appreciate that the Department of Health and Human Services will, of course, have to carry out the law that we pass, and that is your responsibility. Secretary Schweiker came from the Senate and is a very distinguished person. He is doing it and would be expected to do it. I have no quarrel with that. I am not asking that anyone pull punches in terms of the laws passed by the legislative body. You ought to do it, and I know you intend to do it and are doing it.

But on this matter with the bill and what we are trying to do now, we are trying to take these so-called alleged scientific findings and do something with them now in the policy public arena. That is your responsibility; that is the Department of Health and Human Service's responsibility, and I think you do have a responsibility on a whole range of health matters.

The CHAIRMAN. Senator, your 10 minutes are about up.

Senator EAST. Just let me ask one more question, Mr. Chairman, and I will stop. I wonder, Dr. Brandt, in terms of information which you are stressing, and I think rightly so, when the figures show, as they did in the House hearings, that 92 percent of the American people know there are health hazards involved in smoking, what, honestly, else could Government do except to begin to prohibit? I don't even think these gentlemen are prepared to go to those lengths.

I am just asking that the Surgeon General's department stick with general scientific causation, that the Department of Health and Human Services, in the policy public arena, stick with information dimensions of this, and I think we can have a responsible health policy in the United States on smoking or anything else we choose to take up.

So let me rest on that, Mr. Chairman. If they might respond briefly to that, I would appreciate it.

Dr. KOOP. Well, Senator East, I would not take issue with most of the things that you have said and, of all people that you know, I am more anxious than any to be certain that anything that the Surgeon General says in the future carries the absolute utmost credibility, and therefore, if I did not think that this report did that, I would not have allowed it to be issued over my signature.

Let me say that I think your difference and mine, sir, is primarily in one of semantics and in what you consider to be the variables. Being a scientist and not a politician, I would have to assume that the variables in political thought far exceed those in scientific thought, sir. But the ones that——

Senator **East**. But certainly not, for example, in cancer causation.

Dr. **Koop**. Yes, I think that is true because——

Senator **East**. Well, we may be comparing apples and oranges, but variables again, we are both agreeing, is the key concept in establishing scientific causation, and it is intricate, complex, and intractable in so many areas, as in behavioral or physical or natural sciences.

I am only resistant to the notion that our area is more complex than yours. I think, in certain contexts, it is. In other contexts, it might not be. I would want to see a scientific study of the relative complexity of variables in the two fields.

Dr. **Koop**. Let me finish my semantic difference, and that is the difference between cause and mechanism. If we in medical science only treated those diseases for which we knew the mechanism, we would have a much higher mortality for almost everything else we do.

Let me take one of the first diseases in this country that the Public Health Service cared for back in the late 1700's, and that was scurvy. That was before any knowledge of vitamin C. We had no idea of the mechanism of the production of scurvy, but we did know that one of the causes of scurvy was not having any citrus fruits on board ship for long periods of time, and the people who did not have it got scurvy, and when you gave them citrus fruits, they got over their scurvy. So that was cause. The mechanism was totaly unknown.

A more simple explanation: I flick on the light switch all the time, and I know that that causes the light to go on. But I never have understood the flow of electrons along copper wire. That is mechanism.

I think that what Dr. Brandt outlined for you so nicely about the epidemiologists' five points, and which is covered in about the first 40 pages of this report, how we got to that word "cause," I think we have proved the cause of cancer in this particular volume.

Let me also say that when you talk about things like education, I couldn't agree with you more. The actual fact of the matter is that in the polls, between 89 and 92 percent of American people do know that cancer is associated with smoking, but they do not know all of the other things that are associated with smoking.

When you talk to a young person—and remember that I spent my life, the past 40 years of it, with young people—and you talk to a 14-year-old who is about to smoke about cancer when he is 60, he is not the least bit interested. But if you talk to him about what it will do to his wind when he is a runner or what it might do to his lungs when he is 25 or 30, then you have something you can talk with him about.

I would not by any means limit the educational effort in reference to smoking to just cancer. It has got to be far broader than that. With your interest in behavioral sciences, sir, I think you would be very pleased to work along something like this, because what we need is a method of understanding how we can overcome peer pressure in young people who are about to smoke, giving them the facts of health and then working on them psychologically so they don't start, because if you don't start, then you never have to quit.

I could talk on and on about this, sir. I would just conclude by one thing and say that your comments about other substances have a great deal of truth to them, and I would say that I do eat bacon and I do drink drinks with saccharin in them, but I do not smoke.

The **Chairman**. Thank you, doctor. Thank you, Senator East.

Dr. Brandt and Dr. Koop, you have both indicated, at least in Dr. Brandt's statement, that the deaths as a result of smoking are 300,000 a year compared to deaths from all injuries of 105,000. You indicated that smoking is one of three major independent risk factors contributing to coronary heart disease, arteriosclerotic peripheral vascular disease, that smoking is a major cause of cancer of lung, larynx, oral cavity, esophagus; it is a major cause of chronic bronchitis and emphysema; it is a contributing factor in cancer of the urinary bladder, kidney, and pancreas; it is associated with peptic ulcer disease; that maternal cigarette smoking is associated with retarded fetal growth and increased risk of spontaneous abortion and prenatal death and slight impairment of growth and development during early childhood.

You also state that cigarette smoking acts synergistically with oral contraceptives to enhance the probability of coronary and some cerebrovascular disease, that it acts synergistically with alcohol to increase the risk of cancer of the lung and with other risk factors to enhance cardiovascular risk.

Now, that sounds pretty serious to me. Do the health consequences of smoking warrant stronger warning labels on cigarette packages? What do you think, Dr. Koop?

Dr. **Koop**. Well, I think it is broader than just cigarette packages, sir. I think it requires broader education across the whole board. As I indicated to Senator East, I think, if we could stop people from starting rather than stop them from continuing, we would be way ahead. So I think we have got to broaden our whole educational outlook, not just to one aspect.

The **Chairman**. So labels are one aspect of it?

Dr. **Koop**. Certainly.

Senator **Hatch**. And certainly one that ought to be taken.

Senator Packwood.

Senator **Packwood**. I want to ask Dr. Brandt another question. In your testimony before the House, you had this statement: "We believe that the system recommended as proposed section 4(b) might be more effective if all of the proposed warnings appear on each brand simultaneously, so that the smoker does not know which warning may appear on the packages he buys. This is the system in use in Sweden where 16 different warnings appear on packages at a given time. Is that still your position?

Dr. **Brandt**. It is certainly my position.

Senator PACKWOOD. Is it the administration's position?

Dr. BRANDT. I think the issue of precisely how to go about effecting the warnings, stronger warnings, is something that is currently being discussed.

Senator PACKWOOD. Public health warnings are not new. Nathaniel Hawthorne labeled Hester Prynne for the public to watch out for. I think what you are telling me, Dr. Brandt, is this. You are a very good and very true man. You strongly support the labeling; you strongly support, perhaps, at least the Swedish system, and that was in your testimony last week. Between that time and this time, somebody has indirectly talked to somebody in your department and said to tone the testimony down, and you are a good soldier and you have done so, and I am not going to ask you to comment any further on it unless you want to rebut what I am saying.

But it appears to me that the things that you personally believe and support, which were reflected in your testimony last week, touched a sensitive near someplace in the administration, and the testimony today does not reflect quite that staunch support that we saw 5 days ago.

No other questions.

The CHAIRMAN. Thanks. Senator East, do you have any other questions?

Senator EAST. Thanks, Senator Hatch.

I would like to at least give Dr. Brandt the opportunity to respond to the point that I was making earlier, and I had overrun my time.

Not to make you try to answer the unanswerable, and I suppose it will sound a little bit like a rhetorical question, but the polls again indicate, and I think commonsense and conventional observation would indicate that probably the Government's information program on the hazards of smoking has been the most widespread and high profile, high visibility campaign of any health issue in the United States, I would say transcending again the general problems of salt, sugar, nutrition, obesity, high blood pressure, genetic problems, automobile accidents, auto safety, and a whole range of other things that I know you have to deal with—and, of course, the Surgeon General is rightly interested in them—but on this question of informing us, it strikes me that in recent years we have seen that these things tend to feed on themselves. I think this hearing and this bill are an example. We single out certain things, and a certain obsession builds up about them. I think it tends to distort the broad pattern of what makes for good health.

In other words, if someone smoked 5 cigarettes a day, or 10, but yet was obese, was a heavy user of salt, a heavy user of sugar, drank too much, and drove too fast, one might really say, frankly, in a balance concept, their problem was not smoking so much as it was the combination of the other variables.

I am just concerned about this obsession that we take smoking and raise it to the first principle of good health, and everything else is sort of downplayed, and we go at it tooth and nail. I am not too sure that at some point we do not do a disservice to the American people in terms of distorting the whole range of things they should be weighing.

Now, again, getting back to information, I think information of all kinds should be passed out, good science and good information. I simply say, Dr. Brandt, when over 90 percent of the American people know the health hazards of smoking and we can continue to inform them in reasonable and prudent and responsible ways, what else can Government genuinely do, except in the case of Sweden where they have announced that they want to prohibit it? I do not think, in a free society, we are quite prepared to go that far. I am sure you gentlemen are not; I would not be; and there are other areas where I think we should not attempt to prohibit—you know, we are not going to force people to jog; we are not going to force them to cut down on salt, sugar, obesity.

There is something about free choice. Informed people, yes, but they make free choices. I don't know how Government can do more than that and be responsible and be consistent with the history of what we consider to be responsible Government in the Anglo-American tradition. The centralized Socialist approach in Sweden—we are not trying to emulate that in this country. I think to use that as a model is chilling, in and of itself. I had not greatly admired Swedish Government, quite frankly, and I am sure Senator Hatch does not, either.

I just wonder, can we inform them any more than we have, Dr. Brandt?

Dr. BRANDT. I think we must continue to inform them, particularly as new evidence accumulates and new information is available. I think it is our responsibility to do so. I do not think that our preventive programs should be aimed only at cigarette smokers. I think, clearly, that alcohol abuse, drug abuse, the abuse of certain other elements in our society like salt, and so forth, need to be explained and the best scientific evidence brought to the American people so that they can decide on healthy lifestyles.

However, at the same time, I think it is important that we continue these efforts and that we target them at those areas that have the greatest possibility of being prevented, such as infectious diseases and things of that sort where we do target our efforts, and at leading problems. I think that the evidence is absolutely overwhelming now, and this is not something new; this has been going on since the 1930's when the first reports linking cigarette smoking with cancer came out. It just seems that it is clearly the single most preventable cause of human suffering and illness in this country, and I think, therefore, it behooves us to make that a high priority.

I am not arguing that we should make it only cigarette smoking. Obviously, the very thing you are talking about, people who smoke and drink and drive too fast; clearly, all of those things have to be pointed out to them. I do not have any argument that people have to have the right of choice, and I in no way would endorse prohibition of cigarettes because, pragmatically, I do not think it would work, anyway, but I still think it is important.

I think we have been responsible in our efforts to do that. I think this is a responsible scientific document. I think the information that we make available to the public is not intended to terrorize or otherwise; it is intended to inform. I think that we have to continue to inform them. We have to give them the latest kind of information that is available as best we can and in the most effective manner that we can devise.

Senator EAST. So the role of Government would be to inform but not to prohibit, as you are stating it. Would that somewhat accurately present your point of view?

Dr. BRANDT. I am certainly not an advocate of prohibition because, as I say, irrespective of the philosophic viewpoint, I think, pragmatically, it is not going to happen. So I think we have to continue to point out to people the problems that they are having and try to give them ways.

At the same time, I think we have another obligation, and that is that I think we need to continue our research efforts on understanding why people smoke and understanding how we can help people quit smoking, and we will do that, and we will inform them about the results of that research as well.

Senator EAST. Thank you, Dr. Brandt.

The CHAIRMAN. Thank you. We appreciate all three of you coming today, and thank you for taking the time to be with us.

Senator PACKWOOD. If I might say one thing, Mr. Chairman, the Commerce Committee will be having hearings on this on April 26. I assume you may be coming, Dr. Brandt.

Dr. BRANDT. Yes, sir.

Senator PACKWOOD. Maybe the administration would have reached some conclusions by that time.

Dr. BRANDT. Yes, sir.

The CHAIRMAN. Thank you.

Our second panel of witnesses today are physicians who will focus on two scientific aspects of this bill.

The first witness is Dr. William Pollin, the Director of the National Institute on Drug Abuse, and the second witness is Dr. John Holbrook, an associate professor of medicine at the University of Utah College of Medicine. So we are very happy to have both of you scientists with us here today, and we will look forward to taking your testimony at this time. We will start with you, Dr. Pollin.

STATEMENT OF WILLIAM POLLIN, M.D., DIRECTOR, NATIONAL INSTITUTE ON DRUG ABUSE

Dr. POLLIN. Mr. Chairman and members of the committee, thank you for the opportunity to testify on the addictive properties of cigarette smoking.

On the basis of our review of this question and of research conducted in NIDA's own laboratories, it is our conclusion that cigarette smoking represents a prototypic-dependence process and is in fact the most widespread example of drug dependence in this country.

With your permission, Mr. Chairman, I will summarize the evidence for these conclusions and also summarize my prepared statement.

There had been frequent suggestions in the past from both research and policy sources that the question of the possible addictive nature of cigarette smoking needed review. In July 1978, NIDA and a committee of the National Academy of Sciences cosponsored a conference which explored the background of this issue. A copy of the proceedings of that meeting, "Cigarette Smoking as a Dependence Process," has been submitted for the record.

In August 1979, a NIDA-sponsored technical review was held to specifically review the question, "Is cigarette smoking an addiction?" It concluded, and I quote, "Cigarette smoking behavior should be considered a form of addiction, and tobacco in the form of cigarettes, an addicting substance." A summary of that review and the participants has also been submitted.

More recently, the National Advisory Council on Drug Abuse passed the following resolution:

> The National Advisory Council on Drug Abuse strongly recommends to the Surgeon General that words be added to the warnings on cigarette packages. The label should read, "The Surgeon General has determined that cigarette smoking is addictive and dangerous to your health."

Consistent with these conclusions and recommendations is the fact that DSM-III, which is the current standard diagnostic manual of psychiatric disorders in the United States, and ICD-9, the World Health Organization's International Classification of Disease, both include "tobacco dependence" as a drug-dependence disorder.

Thus, five major national and international reviews of this question, which have involved the most knowledgeable and experienced authorities in the area, have all reached the same conclusion: Cigarette smoking is an addiction.

"Dependence" in the classic sense is indicated by: One, persistent regular use of a psychoactive drug; two, attempts to stop such use which lead to discomfort and which often result in termination of the effort to stop; three, continued drug use despite damaging physical and/or psychological consequences; and, four, persistent drug-seeking behavior.

People are drug dependent when a drug takes over and controls their ability to choose freely to take the drug or not. The relative degree of dependence between the two most widely used licit drugs in this country—alcohol and tobacco—is demonstrated by these facts: Whereas the large majority of Americans who use alcohol are both subjectively and objectively able to satisfactorily control their level of use, and only some 10 percent lose control, the opposite is true with tobacco smoking. The great majority of smokers report that they would like to smoke less or quit smoking but find it very difficult or impossible to do so.

Ninety-nine percent of those who say they smoke tobacco smoke regularly. Seventy percent of those who report current smoking say they smoke more than 15 cigarettes a day. Nine out of ten smokers say they would like to quit smoking. Unfortunately, between 80 and 85 percent of current smokers who have ever tried to quit say they have been unable to do so for more than 3 months.

Many researchers are currently exploring the same questions that we ask of any drug-using behavior: What factors determine initial experimentation or use; the progression from casual recreational use to regular, compulsive use; the achievement and maintenance of abstinence; and the factors leading to the high rate of relapse.

The bulk of research findings to date implicate nicotine as the main factor in establishing and maintaining dependence on tobacco. This results from nicotine's powerful biological and psychological effects, which include stimulation of the release of a number of substances—norepinephrine, epinephrine, growth hormone, cortisol, vasopressin, and probably beta endorphin; the production of behavioral arousal and EEG alerting patterns; and the fact that nicotine is one of the most rapidly metabolized of all self-administered substances.

Nicotine is a psychoactive drug; that is, it influences subjective state and behavior. Not all psychoactive drugs lead to dependence or craving: The major tranquilizers, for example, are powerful drugs which do not do so.

NIDA's Addiction Research Center has developed a test, the ARC Inventory, for precisely quantifying psychological and/or subjective drug effects. Over the years, this test has been administered to over 3,000 individuals, both with and without drug abuse histories.

One major finding of current NIDA studies, derived from the euphoria subscale of this ARC Inventory, is the marked similarity it demonstrates between morphine, cocaine, and nicotine. During the first several minutes after administration of these three drugs, there is an immediate, marked, and similar change in feeling state. During this period, morphine, cocaine, and nicotine all feel very much alike to the experimental subject. The subject experiences a euphoric rush which, after a few minutes, however, shows a variable course depending on the particular drug.

Both from self-reported information and from intravenous administration of nicotine, we have found that subjects who have histories of narcotic addiction say that they like tobacco and/or nicotine as well as or even more than other drugs. An important British epidemiologic study of 210 subjects seeking treatment for opiate addiction showed that on a liking scale of 1 to 5, with 5 being the most-liked drug, tobacco received a liking score of 4.3, as compared with 4.7 for heroin, 4.2 for cocaine, and 2.4 for amphetamines.

This same study also measured these same subjects' comparative perceived need, as they described it themselves, for different drugs. On a scale of zero to 4, with 4 representing the most needed, tobacco received the highest score of any of the 10 drugs tested, averaging 3.3, compared to 2.8 for heroin and 1.5 for cocaine.

There may well be questions as to the generalizability of the subjectively felt needs of individuals who are on treatment for drug abuse to the general population. Nonetheless, we think that these data are indicative of a powerful, compulsive effect of tobacco smoking in general and the ingestion of nicotine in particular. These data support both the NIDA ARC studies and also the very common anecdotal reports that heroin addicts find it more difficult to give up tobacco than heroin.

As with other classic drugs of abuse such as the opioids and sedatives, tolerance and physical dependence are important characteristics of a drug because they may exacerbate the user's tendency to continue its use. Both of these are shown to varying extents to be associated with cigarette smoking.

Mr. Chairman, it is likely that drugs such as nicotine and cocaine, which are very powerfully habit forming and yet do not as yet show substantial evidence of being physically addictive, may not do so because we have not yet learned enough about the relationship between brain, drugs, and behavior to be able to identify those physical systems which are at the basis of compulsive and addictive drug use patterns.

The important point which must be stressed in the discussion of psychoactive and addictive drugs is the relative degree of control over the behavior of users which that drug is able to achieve.

We have heard about the severe health consequences that result from smoking. Smoking behavior itself is the disease process which leads to those health consequences. When we can provide more effective help to smokers who wish to regain freedom of choice and reduce or discontinue smoking behavior, then the hundreds of thousands of yearly smoking-related cancer and heart deaths can be sharply reduced.

Thank you, Mr. Chairman. I would be happy to respond to any questions you or members of the committee may have.

[The prepared statement of Dr. Pollin and report of the Task Force on Smoking follow:]

William Pollin, M.D.
Director
National Institute on Drug Abuse

Alcohol, Drug Abuse, and Mental Health Administration
Public Health Service
Department of Health and Human Services

before the

Committee on Labor and Human Resources
United States Senate

on

Tuesday, March 16, 1982

Mr. Chairman and members of the Committee, thank you for the opportunity to testify on the addictive properties of tobacco smoking. On the basis of our review of this question and of research conducted in our own laboratories, it is our conclusion that cigarette smoking represents a prototypic dependence process and is in fact the most widespread example of drug dependence in this country. Let me summarize the evidence for these conclusions.

There had been frequent suggestions in the past from both research and policy sources that the question of the possible addictive nature of cigarette smoking needed review. In July 1978, NIDA and the National Academy of Sciences cosponsored a conference which explored the background of this issue: a copy of the proceedings of that meeting "Cigarette Smoking as a Dependence Process," anda list of participants has been submitted for the record. Finally, in August 1979, a NIDA sponsored Technical Review was held to specifically review the question "Is cigarette smoking an addiction?"; it concluded, and I quote, "Cigarette smoking behavior should be considered a form of addiction, and tobacco in the form of cigarettes, an addicting substance." A summary of that review and the participants, has also been submitted. More recently, the National Advisory Council on Drug Abuse passed the following resolution:

> "The National Advisory Council on Drug Abuse strongly recommends to the Surgeon General that words be added to the warning on cigarette packages. The label should read 'The Surgeon General has determined that cigarette smoking is addictive and dangerous to your health'."

Consistent with these conclusions and recommendations is the fact that DSM-III, the current standard diagnostic manual of psychiatric disorders in the United

States and ICD-9, the World Health Organization's International Classification of Disease, both include "Tobacco Dependence" as a drug dependence disorder. Thus, five major national and international reviews of this question, which have involved the most knowledgeable and experienced authorities in the area, have all reached the same conclusion: cigarette smoking is an addiction.

"Dependence"in the classic sense is indicated by (1) persistent regular use of a drug, (2) attempts to stop such use which lead to discomfort and which often result in termination of the effort to stop, (3) continued drug use despite damaging physical and/or psychological problems, and (4) persistent drug seeking behavior. People are drug dependent when a drug takes over and controls their ability to choose to take the drug or not. The relative degree of dependence between the two most widely used licit drugs in this country--alcohol and tobacco--is demonstrated by the fact that whereas the large majority of Americans who use alcohol are subjectively and objectively able to satisfactorily control their level of use, and only some 10 percent lose control. The opposite is true with tobacco smokers: the great majority of smokers report they would like to smoke less, or quit smoking, but find it very difficult or impossible to do so.

Data from the National Center for Health Statistics tell us that 99 percent of those who say they smoke tobacco, smoke regularly. Seventy percent of those who report current smoking, say they smoke more than 15 cigarettes a day. Nine out of 10 smokers say they would like to quit smoking. Unfortunately, between 80 and 85 percent of current smokers who have ever tried to quit say they have been unable to do so for more than three months. Smokers spend time, money, and a lot of energy on a behavior they would rather not engage in. This view of dependence is consistent with how we use the term "dependence" with other drugs, such as alcohol and cocaine.

Many researchers--some supported by NIDA--are currently exploring the same questions that we ask of any drug-using behavior: what factors determine (1) initial experimentation or use; (2) the progression from casual recreational use to regular, compulsive use; (3) the maintenance of abstinence; and (4) the high rate of relapse.

The bulk of research findings to date implicate nicotine as the main factor in establishing and maintaining dependence on tobacco. This results from nicotine's powerful biological and psychological effects, which include stimulation of the release of a number of substances (norepinephrine, epinephrine, growth hormone, cortisol, vasopressin, and probably beta endorphin); the production of behavioral arousal and EEG alerting patterns; and the fact that it is one of the most rapidly metabolized of all self-administered substances.

Nicotine is a psychoactive drug. That is, it influences subjective state and behavior. Not all psychoactive drugs lead to dependence or craving: the major tranquilizers, for example, are powerful drugs which do not do so. NIDA's Addiction Research Center has developed a test --the ARC Inventory-- for precisely quantifying psychological and/or subjective drug effects. Over the years, this test has been administered to over 3,000 individuals, both with and without drug abuse histories. A major finding of current NIDA studies, derived from this euphoria sub-scale of the ARC Inventory, is the marked similarity that exists between morphine, cocaine, and nicotine. During the first several minutes after administration of the drugs, there is an immediate and marked change in feeling-state (euphoria). This reflects the fact that during this period morphine, cocaine, and nicotine all "feel" very much alike. The subject experiences a "rush," which after a few minutes shows a variable course depending on the particular drug.

Both from self-reported information and from intravenous administration of nicotine, we have found that subjects who have histories of narcotic addiction say they "like" tobacco and/or nicotine as well as or even more than other drugs. A British epidemiological study of 210 subjects seeking treatment for opiate addiction showed that (on a scale of one to five, with five being the "most liked"), tobacco received a "liking" score of 4.3, compared with 4.7 for heroin, 4.2 for cocaine, and 2.4 for amphetamine. This same study measured these subjects'comparative perceived "need" for different drugs. On a scale of 0 to 4, with 4 representing the "most needed", tobacco received the highest score: 3.3--compared to 2.8 for heroin, and 1.5 for cocaine. Although there may be questions as to the generalizability of the felt needs of individuals in treatment for drug abuse to the general population, we think that these data are indicative of the powerful, compulsive effect of tobacco smoking in general and the ingestion of nicotine in particular. These data support both the NIDA ARC studies and the common anecdotal report that heroin addicts find it more difficult to give up tobacco than heroin.

As with other classic drugs of abuse such as the opioids and sedatives, tolerance and physical dependence are important characteristics of a drug because they may exacerbate the user's tendency to continue its use. Tolerance, for instance, reduces the pharmacological effects of drugs and may lead to more frequent administration of higher doses of the drug, which in turn may produce graver health and social consequences for the user. Tolerance has been demonstrated for some of the effects of smoking cigarettes and also to the effects of many of the components of cigarettes. As most of us know, nausea and dizziness is common among novice smokers, but disappears with experience. Metabolic tolerance can be demonstrated in smokers to various components of cigarette smoke (for example, nicotine), as well as to a wide variety of drugs such as barbiturates and chlorpromazine. As with other drugs of abuse, withdrawal signs do appear when heavy smokers abruptly quit. There is some variability in withdrawal symptoms, but it is not unusual for a smoker who stops smoking to show a decrease in excreted epinephrine and norephinephrine and its metabolites. Furthermore, there is a decrease in mean EEG frequency, in heart rate, an increase in appetite and weight, and an impairment in performance on psychomotor tasks and in concentration. Disturbance in sleep may occur and the individuals may feel anxious, irritable and even aggressive. Finally, most individuals who are trying to stop feel an increased craving for tobacco smoking.

Mr. Chairman, it is likely that drugs such as nicotine and cocaine, which are very powerfully habit forming, and yet do not show irrefutable evidence of being physically addictive, may not do so because we have not yet learned enough about the relationship between brain, drugs, and behavior to be able to identify those physical systems which are at the basis of compulsive drug use patterns. The important point which must be stressed in the discussion of psychoactive drugs is the relative degree of control over the behavior of users which that drug is able to achieve. We have heard about the severe health consequences that result from smoking. Smoking behavior itself is the disease process which lead to these health consequences. When we can provide more effective help to smokers who wish to regain freedom of choice and reduce or discontinue smoking behavior, then the hundreds of thousands of yearly smoking-related cancer and heart deaths can be sharply reduced.

Thank you, Mr. Chairman. I would be happy to respond to any questions you may hve at this time.

NATIONAL INSTITUTE ON DRUG ABUSE

TECHNICAL REVIEW ON CIGARETTE SMOKING AS AN ADDICTION

Thursday and Friday, August 23 and 24, 1979

FINAL REPORT

Moderators:

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TECHNICAL REVIEW ON CIGARETTE SMOKING AS AN ADDICTION: REPORT ON THE TASK FORCE ON SMOKING

1. STATEMENT OF THE PROBLEM

Cigarette smoking is the single most important environmental factor contributing to early death and disability in the United States. In 1978, 33 percent of the population, age 17 and over, were current cigarette smokers. Induction into smoking begins in the great majority of individuals before 21 years of age and in over half, before 18 years of age. Despite teenagers' disclaimers that their cigarette smoking can be voluntarily curtailed, the onset of smoking at this age results in a high probability of sustained lifelong use. Of teenagers who smoke more than one or two casual cigarettes, 85 percent escalate to a lifestyle of regular smoking. It is estimated that only 2 percent of smokers consume cigarettes on an occasional basis. In general, the number of cigarettes consumed by smokers averages 30 per day. Each inhaled puff of cigarette smoke delivers a dose of drug to the brain resulting in 50-70,000 such doses per person every year. There is no other form of drug-taking that occurs with such regularity and frequency.

Despite widespread public acknowledgement of the health consequences of smoking and the documented statements made by the great majority of smokers that they would like to quit, a very large number have been unsuccessful in their attempts. More specifically, of the 33 percent of Americans, age 17 and over, who are smokers, three out of four express a desire to quit. Indeed, over 60 percent of these current smokers claim to have made at least one serious attempt. During 1978 alone, over 30 percent (17 million) attempted to quit smoking. Unfortunately, the long-term probability of success on any given quit attempt is only 20-25 percent. Even those who seek professional help and are successful in completing an organized smoking cessation program show a 60-80 percent return to smoking within one to two years. Furthermore, the probability of relapse over time to cigarette smoking shows remarkable similarity to that observed with alcohol and heroin use. It is of interest to note that individuals who use both heroin and cigarettes report that they would find it easier to give up heroin than cigarettes.

2. DEFINITION OF ADDICTION AND AN ADDICTING SUBSTANCE

Before proceeding to consider whether current evidence allows the categorization of cigarette smoking as an addiction, we must define this term. In its broadest sense, addiction is a state characterized by the repeated ingestion of a substance that is toxic and leads to undesirable social consequences. This definition encompasses other terms often used to describe nonmedical drug use, such as abuse, habituation, and dependence. An addicting substance is one that has: (1) pharmacological properties leading to compulsive use; (2) a capability of producing organ and/or behavioral toxicity; and (3) a use pattern associated with adverse social consequences. In addition, this term is generally applied when the ingestion of such substances is viewed by a large segment of the society as undesirable.

3. ANIMAL STUDIES

There have been two general approaches to obtaining data to determine whether tobacco can be viewed as an addicting substance: (1) the study of cigarette smoking per se, and (2) studies of nicotine alone, since its pharmacological actions suggest that it is the most likely component in tobacco leading to its repetitive use. A variety of behavioral studies of the action of nicotine have been carried out in animals. One of the methods used in the animal laboratory to determine the addiction potential of drugs is the drug self-administration procedure. There is a good correspondence between drugs that are self-administered by laboratory animals and those that are common drugs of addiction in humans. This generality holds true for drugs within the opioid, sedative, and psychomotor stimulant class. Furthermore, drugs which are not addictive substances are not self-administered by animals.

Several studies have demonstrated that intravenous nicotine can maintain self-administration behavior in rats and monekys, but the environmental conditions under which this occurs appear more limited than with drugs such as opioids or psychomotor stimulants. Nicotine appears to be a substance which has the pharmacological properties necessary to lead to its repeated ingestion. Doses of nicotine which are otherwise self-administered may even function as punishers to suppress behavior under certain environmental conditions. The existence of these aversive effects of nicotine may account in part for changes in human cigarette smoking after variations in nicotine content or treatment with nicotine or nicotine antagonists. Effects of pharmocological treatment with nicotine antagonists can alter smoking behavior in humans and suppress nicotine self-administration in animals. Clearly, further studies are needed to determine the range of conditions over which nicotine will maintain or suppress behavior, the critical factors controlling these properties of nicotine, and the ways in which pharmacological treatments can alter smoking behavior or self-administration of nicotine.

There has also been limited research on actual cigarette smoking in animals. The majority of monkeys given free access to cigarettes will smoke, but in a rather sporadic fashion. Certain animals, however, will regularly smoke and obtain nicotine blood levels comparable to those obtained by human cigarette smokers.

These studies of smoking behavior and nicotine self-administration in animals support the view that nicotine is the primary constituent in cigarettes that maintains their compulsive use.

4. HUMAN STUDIES

Although it is still not unequivocally clear that nicotine is the only agent responsible for the development of physical dependence on tobacco or the maintenance of smoking behavior, it is certainly the leading contender. Proof of nicotine's primary role still awaits a demonstration that the cigarette withdrawal syndrome is similar to the nicotine withdrawal syndrome. However, there have been several studies implicating nicotine's role in the subjective aspects of smoking as well as the frequency. For instance, subjects have been given cigarettes without nicotine (or with greatly diminished nicotine) and they fail to report their customary enjoyment of smoking. Some subjects seem to enjoy the low-nicotine cigarettes, but it is possible that they are able to extract more nicotine by changing their manner of smoking (e.g., increased inhalation). Another type of experiment involves delivering nicotine to subjects via another route of administration besides inhalation. Subjects of these experiments do not experience the usual degree of satisfaction that they get from their customary cigarettes, although in some instances they do report some satisfaction from the nicotine.

Further evidence implicating the role of nicotine in cigarette smoking is provided by human studies that have directly measured cigarette smoking behavior. These studies have shown: (1) increases or descreases in nicotine dose in cigarettes are associated with compensatory changes in smoking which tend to maintain nicotine blood levels within certain limits; (2) manipulation of urinary excretion of nicotine either upwards or downwards is also associated with compensatory changes in smoking which tend to maintain nicotine blood levels within certain limits; (3) pretreatment with nicotine (intravenously or orally) produces compensatory decreases in smoking; and (4) pretreatment with a nicotine antagonist produces elevation in smoking.

5. PHYSICAL DEPENDENCE AND TOLERANCE

As with other classic drugs of abuse such as the opioids and sedatives, tolerance and physical dependence are important characteristics of a drug because they may exacerbate the user's tendency to continue its use. Tolerance, for instance, reduces the pharmacological effects of drugs and may lead to more frequent administration of higher doses of the drug, which in turn may produce greater risks of toxicity or untoward effects on the user.

Tolerance has been demonstrated for the effects of smoking cigarettes and also to the effects of many of the components of cigarettes. Nausea and dizziness is common among novice smokers, but disappears with experience. Metabolic tolerance can be demonstrated in smokers to various components of cigarette smoke (e.g., nicotine, "tar", benzypyrene, carbon monoxide, other compounds) as well as to a wide variety of drugs such as barbiturates and chlorpromazine. Receptor tolerance can be demonstrated to some extent to certain components of tobacco smoke. For instance, nicotine given intravenously has been shown to have a greater physiological effect upon nonsmokers than on smokers. Similarly, tolerance to behavior, such as activity level, has been demonstrated in a wide variety of animal studies. Behavioral tolerance to nicotine also has been demonstrated in animals, i.e., animals learn to compensate for decrements in performance while under the influence of the drug.

There is also evidence of physical dependence to tobacco. Clear signs of withdrawal appear when heavy smokers abruptly quit, although there appears to be considerable variability in its manifestation. When a smoker stops smoking suddenly, he/she frequently shows a decrease in heart rate, sometimes in blood pressure, and a decrease in excreted epinephrine and norepinephrine and its metabolities. Other endocrinological changes may also occur. Furthermore, there is a decrease in mean EEG frequency, an increase in appetite and weight, and an impairment in performance on psychomotor tasks and in concentration. Disturbances in arousal and sleep may occur; and anxiety, irritability, and aggression increase. Finally, there is an increase in craving for smoking which decreases with time. Despite this reported increase in craving, the extent to which physical dependence on tobacco or nicotine influences the frequency of smoking remains to be determined. Human experiments indicate that, following a period of deprivation, irritability and the probability of smoking increase.

In summary, although experimental findings are limited, it is clear that tolerance and physical dependence do occur with cigarette smoking, but the role they play in the maintenance of smoking remains to be explored.

6. CIGARETTE SMOKING AS AN ADDICTION

It would seem clear from the evidence presented that tobacco smoking produces pharmacological effects which often lead to compulsive use. As stated previously in our definition of addiction, it is necessary to demonstrate that an addicting substance produces organ and/or behavioral toxicity. Although the acute behavioral effects of smoking are mild in comparison to those produced by most addicting substances, the multiple deleterious health effects of cigarette smoking, including mortality and disability and their attendant social consequences, are now well established. Few question that the regular use of tobacco leads to a wide range of organ toxicity.

7. CONCLUSIONS

It was the opinion of the group after reviewing the evidence regarding the compulsive use, the toxicity, and the adverse social consequences, that cigarette smoking behavior should be considered a form of addiction, and tobacco in the form of cigarettes, an addicting substance.

8. IMPLICATIONS REGARDING CIGARETTE SMOKING AS AN ADDICTION

(1) The group concluded that cigarette smoking is an addiction. The broadest implication of this conclusion is that cigarette smoking should now be re-examined in light of the range of policy considerations which are presently considered germane to the classic forms of drug addiction such as addiction to the narocitics, sedatives, stimulants, or alcohol.

(2) Given that cigarette smoking is considered an addiction, it could be contended that it should be viewed as a disease. Such a categorization would allow the application of the methods and conceptual formulations of public health to be applied to the smoking problem.

(3) Basic research efforts should be focused on the analysis of cigarette smoking behavior in humans. Research programs should elucidate the behavioral and pharmacological variables which influence both the maintenance and elimination of cigarette smoking behavior. The role of nicotine in the initiation and maintenance of cigarette smoking should be explored with special attention to other components of smoke that may modulate its effects.

(4) Research on psychosocial influences should not be deemphasized, but projects should be sought which try to evaluate psychosocial and pharmacodynamic influences within the same context.

(5) Basic research efforts should be focused on the analysis of nicotine self-administration in laboratory animals. The range of conditions necessary for the establishment, maintenance, and elimination of nicotine self-administration should be studied.

(6) Research efforts should be directed toward establishing an adequate animal model of cigarette smoking behavior.

(7) Research should be undertaken to establish valid, low-cost, and preferably noninvasive measures of cigarette smoking to be used as outcome measures in clinical treatment research. Examples of such measures include urinary nicotine or expired-air CO levels.

(8) Physiological and psychological changes that occur during repeated administration (tolerance and physical dependence) and upon smoking cessation (withdrawal) should be characterized in detail. The contribution of these changes to the clinical phenomenon of relapse to smoking should be established.

* * * *

This report has summarized the Technical Review on Cigarette Smoking as an Addiction. Additional information about the meeting may be obtained from Dr. Pierre Renault, NIDA Division of Research.

The **Chairman**. Well, thank you, Dr. Pollin.

As I understand it, you need to leave to go to a funeral.

Dr. **Pollin**. That is right.

The **Chairman**. When is the latest you need to leave?

Dr. **Pollin**. I have at least 20 to 25 minutes, Mr. Chairman, that I could stay.

The **Chairman**. Do you have any questions?

Senator **Packwood**. I have only one question. That is, on page 2 of your testimony, where you talk about adding the words "addictive" and "dangerous," are you suggesting that to the present label or to any other additional labels that we may choose to legislate we should add the word "addictive"?

Dr. **Pollin**. The National Advisory Council, which passed that resolution, was suggesting the addition of the word "addictive" to the present label. I would conclude from the nature of their discussion that they would think it would be important to have the addictive warning part of any campaign emphasizing the health risks of smoking cigarettes.

Senator **Packwood**. Is that your personal conclusion, also?

Dr. **Pollin**. Yes, it is.

Senator **Packwood**. Is it the administration's position?

Dr. **Pollin**. As Dr. Brandt indicated today, the administration is strongly in favor of stronger health warnings. The specific mechanisms which may most optimally achieve that are still under consideration.

Senator **Packwood**. Thank you.

The **Chairman**. Dr. Pollin, let me ask you this question. We appreciate your expert testimony, and you have concluded in front of this committee that tobacco is an addicting substance and should be treated as a drug.

In your opinion, why has tobacco escaped being regulated like other drug substances?

Dr. **Pollin**. I am sure that there is a complex political history, and I don't really feel equipped at this point to analyze the balance of forces which has led to, indeed, a very different and unique treatment for tobacco as compared with other substances in this country.

The **Chairman**. You have a distinguished background in behavioral science as well. Do you believe that requiring multiple health warnings is going to be an effective method of public education, and do you believe that these types of warnings will result in less people smoking cigarettes?

Dr. **Pollin**. I believe that knowledge of health consequences, in this as in any other field, is clearly not a simple either/or proposition where people either know it or don't know it. Whenever we are concerned with the reasons that lead to behavior change, we are dealing with gradations and multiple levels of knowledge.

I strongly believe that, as is demonstrated by the American advertising industry, which is perhaps one of the most effective behavior change instruments that man has developed, increasing knowledge, increasing presentation of messages, varied presentation of messages, presentation of health consequence knowledge which alerts an individual because it represents a new message rather than one which has become so well known that it is taken for granted and not attended to, that such principles clearly will make efforts to reduce smoking more effective.

The CHAIRMAN. I was surprised to hear that the National Institute on Drug Abuse's study—or NIDA, as you have been referring to it—found marked similarities among nicotine, cocaine, and morphine addiction. What is NIDA doing to inform our citizens about this important finding?

Dr. POLLIN. NIDA contributes to the overall health information programs of the department. The findings with regard to the dependence-producing properties of nicotine represent rather recent findings. They have been publicized to the scientific community. They have been relayed to the department and, we think, will increasingly become part of the department's overall efforts to counter the health consequences of smoking.

The CHAIRMAN. Thank you.

Senator East, do you have any questions?

Senator EAST. I will be brief because I certainly understand the doctor must leave very shortly.

On this matter that you are testifying about here, this matter of addiction, and I am certainly not for a moment questioning your expertise and scientific understanding of this problem, I get somewhat intrigued, though, with this coming together of science and policy here. But just sticking with science for a moment, this matter of addiction somewhat intrigues me—the addictive quality of things, so to speak; for example, what is addiction and the intensity of addiction to this, that, or the other thing, and possible health consequences coming out of it.

For example, certainly addiction to heroin is an infinitely greater health problem than a person who might have a couple of drinks a day. Alcohol, we know, can be addictive. There can be a certain compulsive pattern of use. Some people might have two drinks a day. Some might even say, at that level, it is helpful to health. There is some scientific evidence that it helps in terms of blood circulation and prevents heart attack. Out beyond two drinks a day, some evidence begins to compound the problem: high blood pressure, and so forth.

But addiction—I guess there is physical and psycholcgical addiction, and this problem of variables again makes it complex, and what that would mean in a given context. What I am resistant to is politicizing too quickly—not that you are, but I think this dumps it into a political context here with tobacco—that we avoid this problem of implying that the drive to do something, whether it might be to smoke or to drink or to eat too much or to gamble—I saw a program the other morning on compulsive gambling—this question of obsession to do something is an interesting problem of human behavior. It has interesting and fascinating scientific implications to it.

What I am concerned about is that we not single out this obsession to do something on a particular commodity or activity, and by noting it can be identified with other forms—you mentioned heroin—that, all of a sudden, we are not suggesting that if I had a child that I learned, at the age of 25, was smoking 10 cigarettes a day or whatever, and then I had another child I learned was smoking heroin, and I set a plague on both your houses because there is the commonality of being addictive, or I understood that one of my children was drinking two beers a day, and the addictive quality of alcohol, you see—the common thread of addiction, I think, could distort, really, if I did not watch my emotions, the genuine concern I might have in terms of the relative health of these individuals.

I am just wondering here, when we begin to talk about the addictive quality of tobacco, that we must understand that, assuming it does exist for purposes of argument, it certainly is not to imply that that would have the same kind of health implications for the individual and society as a whole as a person who, for example, is a heroin addict. I think, again, it would bring science into disrepute because the commonsense of the man or woman in the street would say, "Hey, that won't wash." Why? Because commonsense tells us that a heroin addict is destroyed as an individual. They simply become dysfunctional and ultimately destroy themselves physically and mentally. They are no longer employable, and they have this enormously expensive habit and they go to all kinds of things, prostitution and stealing, and it destroys them.

So the addiction in that case has horrendous implications to it, but certainly we would not want to transfer that into an area, for example, of usage of tobacco, which goes back to the beginning of time—cigarettes, pipe tobacco, and so forth. We have had a long cultural experience with it, and I am just resistant to the notion that, "addiction in this area ought to be looked upon as comparable to the addiction of the alcoholic or the addiction of the heroin addict."

I do not think commonsense would support that. Am I coming through to you that I am a little bit concerned that we state these things with proportion and balance so as not to distort a legitimate health concern we might have about any or all forms of addiction?

Mr. POLLIN. I think your point is well taken, Senator East, and it is very necessary indeed to attempt to be precise when we do make these comparisons.

I think, however, that I would end up at the bottom line at some distance from your position in terms of the relative health consequences, certainly to the Nation if not to the individual, with regard to the comparative addictive properties of the several substances that you were mentioning, alochol, tobacco, and heroin.

As I pointed out, though alcohol does cause a grievous burden to many individuals and to the Nation, our best data suggest that of the large numbers of people who use it with some regularity, approximately 90 percent are able to control its use satisfactorily. Their drinking behavior does not run away with them.

Just the opposite ratio is true with regard to smokers. Well over 90 percent of smokers who have ever begun as a youngster to casually experiment with a few cigarettes go on to a regular pattern of smoking and then, as adults, reach a level of smoking which they, themselves, would strongly wish to be able to decrease or to eliminate, and they find themselves unable to do so.

And so, in terms of the comparative addictive strength, if you will, of these three substances, I would say that tobacco is much closer to heroin. Some of the empirical, quantitative data which we are now beginning to assemble is rather startling in terms of self-reports from heroin addicts themselves, who do say that, in the one study, they feel they need cigarettes more than they need heroin or any other drug and who give a liking score to intravenous nicotine that is in the same range as the liking score that they give for heroin and cocaine.

Finally, in terms of the impact on the Nation, it is true that heroin has the capacity to be extremely destructive both to the heroin addict and, where there is a high prevalence of heroin abuse in a given community, to whole communities.

On the other hand, there were a total of approximately 1.9 million deaths due to all causes in this country last year; 300,000 of these is the number of premature deaths accepted by the great majority of scientists who have looked at the question of the health consequences and the mortality and morbidity associated with smoking. That is close to approximately one-sixth, a little bit less than one-sixth of the total deaths from all causes in this Nation last year.

The total number of heroin addicts, our best estimate at this point is approximately 475,000. So, in terms of the hundreds of thousands and millions of people involved, the addictive property of nicotine at this point, it seems to me, should be a greater or at least as great a cause for public concern as the addictive property of heroin.

Senator EAST. Knowing that you must leave quickly, let me focus in on this one more time.

It was my understanding—and I could be in error, but this is what I have been led to believe; you hear it so much from those in the public health field—that the most abused drug in America is alcohol. That is the most abused drug, and it is an addictive drug, as you have suggested. Maryland, for example, recently passed an infinitely tighter law dealing with deaths deriving out of drunken driving.

What I am resisting to the notion of now is, you are singling out from the pack drinking and saying that it is all right, and 90 percent of the people can give it up anyway, and it is no big problem, really, and lumping tobacco in with heroin.

Now, I would submit, in the real world of my commonsense observation of public health problems in America today, that alcohol, in terms of the abuse of it and deaths coming not only from alcoholism and broken families and broken lives and broken homes and automobile accidents, and the widespread use of alcohol and drugs of a worse ilk in high schools, and so on and so forth—and maybe this depends on perspective, but I am profoundly resistant to the notion that that is not really a problem or is less a problem than cigarette smoking.

All I am asking for is a little balance in this whole thing, and I will be darned if I can come to the conclusion that you are implying, it seems to me: Don't worry about the drinking. The real problem we have got in America is smoking." I just cannot buy that as a matter of scientific observation in terms of the impact that one versus the other has had and is having in the public arena. At least people who smoke cigarettes are not killing other people in their automobiles like drunken drivers are. At least it has one merit in a free society: To the extent there is harm, it is self-inflicted.

I just do not want the record to show that I sat here silent while it was said, "Don't worry about the drinkers; the problem you need to worry about, Senators, is the smokers." I am not sure that is true.

Dr. **Pollin**. Well, Senator East, if I conveyed the impression that I thought that alcohol was not a major problem or that one need not worry about it and needed only to focus on cigarette smoking, then I misspoke, and I would like to correct that impression.

What I was trying to convey was specifically comparing the addictive power, the ability of the one substance to control behavior as compared to the ability of the other substance.

With regard to comparative health consequences, the Surgeon General's last report on alcohol and health indicated that there are varying estimates of the number of deaths which may be related to the abuse of alcohol. The maximum figure which has ever been cited is 200,000 deaths per year, and that includes deaths due to automobile accidents, deaths due to careless setting of fires, and so forth.

You are entirely correct in that alcohol has a variety of destructive consequences in terms of destroying family life, individuals' ability to function, and the like, which is certainly not shared by cigarette smoking. But if we precisely try to compare those variables which are comparable—numbers of deaths which most people believe are associated—then, indeed, the numbers of deaths which are associated with cigarette smoking are substantially greater than the number of deaths which, at the most liberal estimate, have been associated with the abuse of alcohol.

Senator **East**. My time is up, Mr. Chairman, and I appreciate that he must go.

The **Chairman**. Thank you, Senator East.

Dr. Pollin, thank you for being here today. We appreciate your testimony.

Dr. **Pollin**. Thank you.

Senator **East**. Thank you.

The **Chairman**. Our next witness will be Dr. John Holbrook, who is a distinguished member of the faculty of the College of Medicine from my home State. John is widely respected for his knowledge of the health-related problems of smoking. He has consulted with the Federal Government for years regarding smoking and health. I am informed by a lot of young people out there that he is one of the best teachers in the medical school, and he wins numerous awards continuously in that regard.

So, John, we are happy to have you here today, and we will look forward to taking your testimony at this time.

STATEMENT OF JOHN H. HOLBROOK, M.D., ASSOCIATE PROFESSOR, UNIVERSITY OF UTAH COLLEGE OF MEDICINE

Dr. **Holbrook**. Mr. Chairman, it is a pleasure for me to speak this morning about tobacco additives.

The Surgeon General's 1981 report, the Changing Cigarette, focused attention on the dramatic increase in consumption of lower tar and nicotine cigarettes, which currently account for approximately 50 percent of the U.S. market.

The reduction in tar and nicotine has been achieved by changes in cultivating and processing of tobacco and in the manufacturing of cigarettes. In this process different classes of substances may be used, including: Flavoring agents, binders, humectants, tobacco extenders, burn-rate accelerators and retardants, artificial tobacco substitutes, fertilizers, and agricultural chemicals.

In the 1981 report, Dr. Julius Richmond, the then-Surgeon General, summarized his concern about the lower tar and nicotine cigarettes as follows:

> A final question is unresolved, whether the new cigarettes being produced today introduce new risks through their design, filtering mechanisms, tobacco ingredients, or additives. The chief concern is additives. The Public Health Service has been unable to assess the relative risks of cigarette additives because information was not available from manufacturers as to what these additives are.

Mr. Chairman, in the highly competitive world of cigarette marketing, flavor additives appear to have been used increasingly to stimulate consumer acceptance of a product containing reduced amounts of tobacco. These additives are not regulated by any agency of the Federal Government, and public disclosure of substances currently in use is not required.

To my knowledge, this is a unique legal loophole. In other words, other industries whose products are taken into the body are regulated, and manufacturers are held responsible for the effects of their products. It should be emphasized that the cigarette is a delivery vehicle which results in the prompt distribution of thousands of tobacco smoke constituents throughout the body.

The evidence concerning the harmful effects of cigarette smoking is compelling: its toxicity, its carcinogenicity, its mutagenicity, and possibly its teratogenicity; in other words, its possible birth defect-producing effects.

What additional effects tobacco additives and also the combustion products of these tobacco additives may exert on the chemical composition and biological activity of cigarette smoke are simply not known. Their presence in tobacco products represents an unmeasured risk—an unmeasured risk for the active smoker, for the passive smoker, meaning those of us in a confined space who are forced to inhale the smoke coming from a cigarette, and for the unborn child. I would emphasize, we simply do not know the potential effects, and it is an unmeasured risk.

There are cogent reasons to anticipate adverse interactions between tobacco additives and other smoke constituents. For example, smokers who are exposed to certain medications—oral contraceptives, for example—and to certain workplace substances—asbestos—face greatly increased health risks compared to both nonsmokers and smokers who are free from such exposures.

In 1972, the R. J. Reynolds Tobacco Co., published a booklet listing several hundred chemical substances or natural products used in the "compounding of tobacco flavors." The classes included several major groups of chemicals, and I will not bother to list these; they are included in my written testimony.

The Hunter committee's second report listed a few hundred substances approved for use as tobacco additives in the United Kingdom. Unfortunately, Mr. Chairman, these lists do not provide specific information on the types and amounts of substances actually being added to tobacco products.

I'll conclude by pointing out that many smokers of lower tar and nicotine cigarettes believe that as long as they use the lower tar and nicotine cigarettes this habit is not harmful. S. 1929 will focus attention on the risk of smoking in general and on the potential risks posed by tobacco additives. This bill will also mandate disclosure of tobacco additives which, in my judgment, is a first step in the assessment of health risks. I applaud and support this bill.

[The prepared statement of Dr. Holbrook follows:]

TESTIMONY BEFORE THE UNITED STATES SENATE LABOR AND HUMAN RESOURCES COMMITTEE ON:

Tobacco Additives

by

John H. Holbrook, M.D.

March 16, 1982

The Surgeon General's 1981 Report, The Changing Cigarette, focused attention on the dramatic increase in consumption of lower "tar" and nicotine cigarettes, which currently account for approximately 50% of the United States market.

The reduction in "tar" and nicotine has been achieved by changes in cultivating and processing of tobacco and in the manufacturing of cigarettes. In this process different classes of substances may be used including: flavoring agents, binders, humectants, tobacco extenders, burn-rate accelerators and retardants, artificial tobacco substitutes, fertilizers, and agricultural chemicals. In the 1981 Report Dr. Julius Richmond expressed concern about the lower "tar" and nicotine cigarettes:

> "A final question is unresolved, whether the new cigarettes being produced today introduce new risks through their design, filtering mechanisms, tobacco

ingredients, or additives. The chief concern is additives. The Public Health Service has been unable to assess the relative risks of cigarette additives because information was not available from manufacturers as to what these additives are".[1]

In the highly competitive world of cigarette marketing, flavor additives appear to have been used increasingly to stimulate consumer acceptance of a product containing reduced amounts of tobacco. These additives are not regulated by any agency of the federal government, and public disclosure of substances currently in use is not required. To my knowledge this is a unique legal loophole, i.e. other industries whose products are taken into the body are regulated and manufacturers are held responsible for the effects of their products. It should be emphasized that the cigarette is a delivery vehicle which results in the prompt distribution of thousands of tobacco smoke constituents throughout the body.

The evidence concerning the harmful effects of cigarette smoke is compelling, e.g. toxicity, carcinogenicity, mutagenicity, and possibly teratogenicity. What additional effects tobacco additives and their combustion products may exert on the chemical composition and biological activity of smoke are not known. Their presence in tobacco products represents an unmeasured risk for the active smoker, the involuntary smoker and the unborn child.

There are cogent reasons to anticipate adverse interactions between tobacco additives and other smoke constituents. For example, smokers who are exposed to certain medications, e.g. oral contraceptives and to certain workplace substances, e.g. asbestos, face greatly increased health risks compared to both nonsmokers and smokers who are free from such exposures.

In 1972 the R.J. Reynolds Tobacco Company published a booklet listing several hundred chemical substances or natural products "used in the compounding of tobacco flavors".[2] The classes included organic acids, amino acids, alcohols, aldehydes, acetals, amides, amines, anhydrides, esters, ethers, pyrones, imides, ketones, lactones, phenols, pyridines, quinolones, indoles, pyrazines, pyrroles, sulfur compounds, herbs, essential oils and extracts. The Hunter Committee's Second Report listed a few hundred substances approved for use as tobacco additives in the United Kingdom.[3] Unfortunately, these lists do not provide specific information on the types and amounts of substances actually being added to tobacco products.

Many smokers believe that smoking lower "tar" and nicotine cigarettes is not harmful. Senate Bill 1929 will focus attention on the risks of smoking in general and on the potential risks posed by tobacco additives. This bill will also mandate disclosure of tobacco additives, which is a first step in the assessment of health risks. I applaud and support this bill.

References

1. U.S. Department of Health and Human Services: The Health Consequences of Smoking - The Changing Cigarette. DHHS. Publication No. (PHS) 81-50156, 1981.

2. Leffingwell JC et al: Tobacco Flavoring for Smoking Products. R.J. Reynolds Tobacco Company, Winston-Salem, North Carolina, 1972.

3. Hunter Report: Developments in Tobacco Products and the Possibility of "Lower-Risk" Cigarettes. Her Majesty's Stationary Office, London, 1979.

The **Chairman**. Thank you, doctor, for your testimony.

You have indicated that although there is no current requirement for tobacco companies to inform the public about chemical additives, you already have knowledge about many of the substances that are being used, and you list them in your statement here today.

Now, why do you think it is important to legislate tobacco companies so that they divulge the nature and amount of additives being used?

Dr. **Holbrook**. If I conveyed the impression that we have specific knowledge about the substances, I did not mean to do that. I simply meant to indicate that lists have been published by tobacco companies of substances that may be used or have been used, and another list is available from the British industry indicating substances that the Government and the tobacco industry in Great Britain have agreed upon. However, we have no precise information concerning substances or amounts of substances, and that is the issue that I would like to leave before this committee today.

I think it is a scientific question. I think scientists should be pursuing the possible interactions between tobacco smoke and additives and the effects these interactions may have on the smoker.

The **Chairman**. Apparently, the United Kingdom has the current knowledge about chemicals being used in the manufacture of cigarettes. Have they done any scientific research in that area, and do we have any benefit of that, or can you cite any information along those lines?

Dr. **Holbrook**. This particular Hunter committee met intermittently over a period of several years, and there was some agreement about substances which would be acceptable. However, my understanding is that some of the substances were grandfathered onto the list; that is, because they were being used, it was considered acceptable to continue their use. However, to my knowledge, precise assessment of the scientific risks, of these additives was not carried out in Great Britain and certainly had not been carried out in our country.

The **Chairman**. I see. Are chemical additives only being used in low tar cigarettes, or are there chemical additives in any type of cigarette?

Dr. **Holbrook**. Well, I think you have to specify what kind of additives you are speaking about. There are different categories of additives—additives used in the processing of tobacco, the growing of tobacco, the manufacturing of cigarettes.

For example, glycerin is a substance used in the manufacturing of cigarettes.

In the intense, burning heat of the cigarette—and I compare a cigarette to a small chemical factory— a substance such as glycerin or, for that matter, cocoa, can undergo profound chemical changes. It is pyrolytic process.

Glycerin, for example, is converted to a substance called acrolein, which is a very potent irritant in the lungs. Cocoa, for example, when it is burned in the intense heat of a burning cigarette, produces many of the substances in its vapor phase which are present in cigarette smoke.

The **Chairman**. We have asked tobacco companies, in this legislation, to include carbon monoxide levels on the cigarette packages. Why is that important?

Dr. **Holbrook**. Carbon monoxide is one of the three substances which, for years, the Surgeon General has identified, along with tar and nicotine, as one of the most toxic substances in cigarette smoke. It is an odorless, colorless gas which excerts its toxic effect through combining with hemoglobin. Hemoglobin is the substance that transports oxygen to cells. Carbon monoxide combines very avidly with hemoglobin and prevents hemoglobin from transporting oxygen to the cell.

Consequently, most cigarette smokers are in a chronic state of low-grade carbon monoxide intoxication. For example, most nonsmokers have 1 percent or less carbon monoxide in their blood; most cigarette smokers have 5 percent carbon monoxide and up to as high as 15 to 20 percent carbon monoxide in their blood. That translates to a decreased oxygen-carrying capacity for the body. This is important in two ways. For people who already have cardiopulmonary problems or for pregnant women, it exacerbates their disease, and in the case of the pregnant woman, it may be the substance that results in smaller babies being born to women who smoke during pregnancy. In terms of the pathogenesis of tobacco-related diseases, carbon monoxide may play a critical role in the development of arterial narrowing.

The **Chairman**. Thank you.

Senator Packwood.

Senator **Packwood**. No questions.

The **Chairman**. Senator East.

Senator **East**. I would just like to, first of all, thank the doctor for coming. Senator Hatch is always bringing in very fine witnesses from his great State of Utah. I remember, we had the attorney general of Utah here not long ago on another subject.

Doctor, on this matter of additives, you rightly suggest, and I would agree with you, that it is an important problem of science and a difficult one which does not yield up its mysteries easily in terms of the need for extensive testing, and so forth, and I would agree with you on that.

I wanted to ask, on the problem of carbon monoxide, not as an expert in physical or medical science, in terms of being able to measure carbon monoxide levels, how perfected, as a matter of science, are we in that area? I am just inquiring as to how far one might go in measuring that if one decided to do that.

Dr. **Holbrook**. Senator East, that is a complicated question, and I am not qualified to discuss the precise technology of how carbon monoxide is measured, but I can tell you about some of the variables that are involved.

There are excellent machines and devices available to measure carbon monoxide, but these measurements are confounded by other variables—such as traveling on congested freeways and workplace exposures. Nonetheless, in spite of these variables, one can obtain reproducible, dependable measurements of carboxyhemoglobin, which is the substance with which we are most concerned.

Those individuals who have elevated levels of carboxyhemoglobin face increased health problems, and cigarette smoking is the most common cause of chronic, low-grade elevation of carboxyhemoglobin.

As a matter of fact, cigarette smokers compensate in a very interesting way for these elevated carboxyhemoglobin levels, by increasing the number of red cells that their bone marrow makes. The body assesses the fact that the oxygen-carrying capacity is reduced and, in a way, it attempts to compensate for this.

Senator **East**. I appreciate your comment on that. The matter here that I would just simply like to raise for the record and bring to the attention of the Chair is that we make sure that if there are burdens of this kind imposed upon the tobacco industry as regards to additives that we might make sure that we not impose burdens upon them that the state of science, on that particular thing, will not permit them to do it; we lack the scientific knowledge on that particular point to do it.

Again, I think we bring the law and science into a bad union there and bring both into disrepute. But it is not to gainsay your point that we ought to always be willing in this or, I would say, in any other area, to pursue the relative correlations of additives and what their general health impact is on the individual or society as a whole. I again do not want our good intentions to get ahead of what our scientific knowledge will allow us to do in this or any other area.

Dr. **Holbrook**. Senator, I think I can reassure you, this is a routine measurement performed in many community hospitals.

Senator **East**. But, as you have indicated, the difficulty with it is that—again, back to our problem, as we have noted at the outset with Dr. Koop—it is a problem of variables and isolating them and determining precisely the health impact upon the individual or society as a whole, then. It is not simple; it is intricate and complex. Our control of all the variables, we have to acknowledge, is not there, so we are going a great deal on supposition.

I just want to make sure that, in the governmental policy, public policy arena, we do not let our desire to do something get away from what our scientific knowledge at this point will sustain. Let us continue to probe the scientific correlations in the laboratories, release that information, and do the best we can with it but not force it beyond what the traffic will bear at the moment.

I found your testimony helpful to remind us of that. Thank you, Mr. Chairman.

The **Chairman**. Thank you, Senator East. Thanks, Dr. Holbrook. We appreciated your testimony.

Our next witness is Dr. Charles LeMaistre, a distinguished scientist and physician who is currently serving in two major positions. He is president of the University of Texas, M. D. Anderson Hospital and Tumor Institute, and he is also representing the Coalition on Smoking or Health, a large group of voluntary and private agencies who have determined to work together in smoking prevention education.

So, Dr. LeMaistre, it is good to see you again and nice to have you here. You may proceed with your testimony.

STATEMENT OF CHARLES A. LeMAISTRE, M.D., PRESIDENT, UNIVERSITY OF TEXAS M. D. ANDERSON HOSPITAL AND TUMOR INSTITUTE, ON BEHALF OF COALITION ON SMOKING OR HEALTH

Dr. **LeMaistre**. Thank you very much, Mr. Chairman.

Mr. Chairman, members of the committee, Senator Packwood, I am Charles LeMaistre, president of the University of Texas M. D. Anderson Hospital and Tumor Institute. Today I testify in support of Senate bill 1929 on behalf of the American Cancer Society, the American Lung Association, and the American Heart Association, plus 27 other health, education, and youth leadership groups. This is the newly organized Coalition on Smoking or Health.

Our Nation's most prestigious agencies have banded together here in Washington and throughout the Nation out of concern for the tragic consequences of smoking to Americans. A statement describing the coalition's blueprint for action is submitted for your record. The blueprint explains that the coalition's primary emphasis is smoking prevention through education.

Because of the uniting of the concerned private sector under the banner of this coalition and the tremendous public impact of the most recent Surgeon General's report on "The Health Consequences of Smoking," the legislation before this committee could not be more timely. In particular, we commend the thrust of this bill to provide information not now available and thereby improve the ability of the American public to make an informed choice on the decision to smoke or not to smoke. These decisions, especially those made by younger Americans, are of critical importance to the health of our Nation.

You have already heard this morning and previously, I am sure, about the consequences of past poorly informed decisions; they are quite familiar. A 1981 estimate of the Department of Health and Human Services states the results very succinctly: 340,000 premature American deaths annually—roughly the population of the capital city of the State of Texas, Austin—health injury to 10 million Americans annually; an annual cost of $41 billion in medical care expenditures and lost economic productivity.

Can there be any reasonable doubt about the stark fact that cigarette smoking is the single most preventable cause of illness and the single most preventable cause of death in the United States? Thirty thousand studies now link cigarette smoking to illness. Six Surgeons General have repeatedly documented the overwhelming evidence that cigarette smoking causes cancer of the lung, the larynx, the esophagus, urinary bladder, mouth, and pancreas; that cigarette smoking causes emphysema and chronic bronchitis; that cigarette smoking is a major risk factor for heart disease; that cigarette smoking is associated with retarded fetal growth and increased risk for miscarriage and prenatal death.

Even so, the message is still not reaching some Americans. The point was made earlier that many smokers are aware in general of the hazards of cigarette smoking, but the Federal Trade Commission's staff report in May of 1981 pointed out, and I quote in part: "Many smokers are unaware of the existence of the relationship between smoking and some of its most serious and widespread health consequences such as heart disease."

Fifty-four million living Americans have already made the decision to smoke cigarettes and continue to smoke today. They are destined to become tomorrow's medical statistics and economic loss. Tragically, a substantial number of Americans are still not fully aware of the dangers of cigarette smoking and take up the habit ignorant of the facts. If the 54 million Americans had never taken up the habit, as you have already heard, approximately 30 percent of all cancer would not occur, and cardiovascular and pulmonary disease would have their toll substantially lessened.

The provisions of this bill for rotational warning labels is a key element in making available to the American public brief, direct and fresh factual information necessary for informed decisionmaking. The present warning label has lost its effectiveness due to its generalized message and sameness over the years.

Perhaps the most important provision of this bill is that which would make it unlawful to manufacture, import, or package for sale any cigarettes without disclosure of all chemicals and substances used in production. For reasons that the nonsmoking majority of Americans find hard to understand, cigarettes remain one of the few substances consumed internally which are exempt from full disclosure of ingredients. Yet the facts are clear: Cigarettes are burned and the products inhaled and taken internally with the result being the designation of cigarettes as the single most preventable cause of illness.

Mr. Chairman, it seems certain that the provisions requiring full disclosure of ingredients including flavoring additives will be attacked as excessive regulation or paternalistic. Quite the opposite has been true, as history clearly reveals.

The Food and Drug Administration has neatly avoided responsibility by ruling that tobacco and tobacco products are neither foods nor drugs and are therefore not subject to the Food, Drug, and Cosmetic Acts.

The Consumer Product Safety Act specifically exempts, by statute, tobacco and tobacco products from the regulations of the act.

The Federal Hazardous Substances Act exempts tobacco and tobacco products from the hazardous products provisions of the act.

The Toxic Substances Act does not provide for tobacco and tobacco products regulation.

One might more properly raise the question: Why the special treatment of these dangerous substances, tobacco and tobacco products? Mr. Chairman, the opportunity for responsible, voluntary disclosure has been present for many years. The only feasible course left for Congress is to require these disclosure provisions in view of the overwhelming evidence that tobacco and tobacco products are not only harmful but life threatening.

The final provision, the statutory authority for the Interagency Council in this bill and in the House for the Office of Smoking and Health, is commendable. This will insure the public is provided both facts and documented evidence in the future. Equally important, this provision will insure the continued monitoring of voluminous data on smoking and health trends, which is most important in keeping our factual information up to date.

The sustaining of these functions is considered an essential first step in the preparation of Americans to make informed decisions about smoking.

Mr. Chairman, this legislation seeks only to provide the consumer with information that the consumer has every right to know. We commend the passage of this bill to assure that the consumer receives the needed information on the tragic consequences of cigarette smoking.

The Coalition on Smoking or Health and the millions of volunteers these private sector groups represent stand ready to work closely with the Congress and government at all levels to see that the information on the hazards of smoking reaches all segments of the American public.

Thank you very much, Mr. Chairman.

[The prepared statement of Dr. LeMaistre with attachments follows:]

Testimony of Charles A. LeMaistre, M.D.
On Behalf of the Coalition on Smoking OR Health
Before the Committee on Labor and Human Resources
United States Senate

March 16, 1982

Mr. Chairman and members of the Committee, I am Dr. Charles A. LeMaistre, President of The University of Texas M. D. Anderson Hospital and Tumor Institute. I testify in support of S.1929 on behalf of the American Cancer Society, the American Heart Association, and the American Lung Association and 27 other health education and youth leadership groups - the newly organized, Coalition on Smoking OR Health. Our nation's most prestigious agencies have banded together here in Washington and throughout the nation out of concern for the tragic consequences to Americans of smoking. A statement describing the coalition's blueprint for action in the cause of smoking prevention education is submitted for your record.

Because of the uniting of the concerned private sector under the banner of the coalition and the tremendous public impact of the most recent surgeon general's report on The Health Consequences of Smoking, the legislation before this committee could not be more timely. In particular, we commend the thrust of S.1929 to provide information not now available to the American public on the health consequences of smoking and thereby improve the ability to make an informed choice on the decision to smoke or not to smoke. These decisions, especially those to be made by younger Americans, are of critical importance to the health of our nation.

The consequences of past poorly informed decisions are quite familiar:

- 340,000 premature American deaths each year due to smoking,
- health injury due to smoking to 10 million Americans,

and,

- a cost to the American public of $41 billion in medical care expenditures and lost economic productivity.

Can there by any question about the stark fact that cigarette smoking is the single most preventable cause of illness and the single most preventable cause of death in the United States? Thirty thousand studies link cigarette smoking to illness; six surgeon generals have repeatedly documented the overwhelming scientific evidence that cigarette smoking causes cancer of the lung, larynx, esophagus, urinary bladder, mouth and pancreas, that cigarette smoking causes emphysema and chronic bronchitis, that cigarette smoking is associated with retarded fetal growth and increased risk for miscarriage and prenatal death. Even so, the message is still not reaching many Americans. 54 million living Americans have already made the decision to smoke cigarettes and continue to smoke today. They are destined to become tomorrow's medical statistics and economic loss. Tragically, a substantial number of Americans are still not fully aware of the dangers of smoking, and take up the habit ignorant of the facts. If the 54 million Americans had never taken up the habit approximately 30% of all cancer would not occur, and cardiovascular and pulmonary disease would have their toll substantially lessened.

The provisions of S.1929 for rotational warning labels is a key element for making available to the American public brief, direct and fresh factual information necessary for informed decision-making. The present warning label has lost its effectiveness due to its generalized message and sameness over the years.

Perhaps the most important provision is that which would make it unlawful to manufacture, import or package for sale any cigarettes without disclosure of all chemicalsand substances used in production. For reasons that the non-smoking majority of Americans find hard to understand, cigarettes remain one of the few substances consumed internally which are exempt from full disclosure of ingredients. Yet the facts are clear: cigarettes are burned and the products inhaled and taken internally with the result being the designation of cigarettes as the single most preventable cause of illness.

Mr. Chairman, it seems certain that the disclosure provisions requiring full disclosure of ingredients including flavoring additives will be attacked as excessive regulation or paternalistic. Quite the opposite has been true as history clearly reveals:

The Food and Drug Administration has neatly avoided responsibility by ruling that tobacco and tobacco products are neither 'foods' nor 'drugs' and therefore not subject to the Food, Drug and Cosmetic Acts.

The Consumer Product Safety Act specifically exempts by statute tobacco and tobacco products from the regulations of the Act.

The Federal Hazardous Substances Act exempts tobacco and tobacco products from the hazardous product provisions of the Act.

The Toxic Substances Act does not provide for tobacco and tobacco products regulation.

One might more properly raise the question: why the special treatment of these dangerous substances, tobacco and tobacco products? Mr. Chairman, the opportunity for responsible, voluntary disclosure has been present for many years. The only feasible course left is for Congress to require these disclosure provisions in view of the overwhelming evidence that tobacco and tobacco products are not only harmful, but life threatening.

The provision of statutory authority for the Office of Smoking and Health is commendable and will provide the public with both facts and documented evidence. Equally important, this provision will ensure the continued monitoring of the voluminous data on smoking and health trends. The sustaining of these functions through stability of the Office of Smoking and Health is considered an essential first step in the preparation of Americans to make informed decisions about smoking.

Mr. Chairman, this legislation seeks only to provide the consumer with information that the consumer has every right to know. We commend the passage of S.1929 to assure that the consumer receives the needed information on the tragic consequences of cigarette smoking. The Coalition on Smoking OR Health and the millions of volunteers these citizen groups represent, stand ready to work closely with the Congress and government at all levels to see that information on the hazards of smoking reaches all segments of the American public.

APPENDIX TO
TESTIMONY OF CHARLES A. LEMAISTRE, M.D.
ON BEHALF OF THE COALITION ON SMOKING OR HEALTH
BEFORE THE COMMITTEE ON LABOR AND HUMAN RESOURCES
UNITED STATES SENATE

MARCH 16, 1982

Coalition on Smoking OR Health
A PUBLIC POLICY PROJECT WITH THE
NATIONAL INTERAGENCY COUNCIL ON SMOKING AND HEALTH

STEERING COMMITTEE
John D. Kessler, Chairman
American Heart Association
Alan C. Davis
American Cancer Society
Robert G. Weymueller
American Lung Association
Robert M. Dougherty, Jr., M.D., Ph.D.
Chairman, National Interagency
Council on Smoking and Health

March 5, 1982

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COALTION ON SMOKING OR HEALTH SEEKS TO INFLUENCE LEGISLATORS

WASHINGTON, March 5 -- A new "Coalition on Smoking OR Health" backed by [illegible]rty of the nation's leading health, education and youth leadership organizations, has been formed to advance the cause of smoking prevention in the United States, it was announced today.

The coalition, which will be based in Washington, D.C., will bring smoking prevention issues to the attention to legislators and federal officials and will promote support for federal smoking prevention education programs, according to John D. Kessler, chairman of the coalition's steering committee.

"The Coalition on Smoking OR Health serves as the public policy action arm of the National Interagency Council on Smoking and Health (NICSH)," Kessler said. NICSH is a voluntary association of leading groups formed in 1967 to inform the public about the dangers of cigarette smoking. The coalition will work closely with the three major voluntary health organizations -- the American Cancer Society, the American Heart Association and the American Lung Association.

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"CIGARETTE SMOKING IS THE SINGLE MOST PREVENTABLE CAUSE OF DEATH IN THE UNITED STATES"

Kessler said the coalition will immediately focus on promoting a substantial increase in the federal excise tax on cigarettes to deter children and youth from starting to smoke and would support enactment of a Comprehensive Smoking Prevention Education Act (H.R. 4957 and S. 1929) that would, among other provisions, provide for strengthened cigarette warning labels.

He cited other immediate public policy priorities: "We are raising the question of adequate federal protections for Americans against exposure to toxins in tobacco, which is now exempt from protective controls which govern foods, drugs, substances and products.

"And we are questioning the consistency and propriety of national public policies which allocate tax dollars to inform Americans about the health dangers of cigarette smoking while at the same time allocating additional tax dollars to subsidize the production of tobacco."

Robert M. Daugherty, Jr., M.D., Ph.D., chairman of the NICSH said, "We welcome the establishment of the Coalition on Smoking OR Health as a means by which we can better encourage federal initiatives and activities on smoking and health." Dr. Daugherty is dean of the school of medicine at the University of Nevada at Reno.

"Although the prevalence of smoking is declining," Dr. Daugherty said, "we do not underestimate the difficulty of the task that lies ahead." He added, "The tobacco industry, with its vast resources, is very good at waging campaigns designed to create diversion and doubt in the minds of the public."

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"To counter the immense determination of the tobacco industry to keep Americans smoking, our limited resources must be effectively coordinated and the interface with the federal sector maintained," he said. "We hope the coalition will serve to focus our efforts in this critical public health area."

Kessler said the coalition hoped to counter fallacies, myths and misinformation on the health hazards of smoking. In 1981 the Department of Health and Human Services estimated that cigarette smoking caused 340,000 premature deaths, injured the health of an estimated 10 million Americans and cost $41 billion in medical care and economic productivity.

"As the Surgeon General's Report on the Health Consequences of Smoking pointed out," Kessler said, "cigarette smoking is the chief, single avoidable cause of death in our society and the most important health issue of our time."

"We agree with the Surgeon General and we believe that the majority of Americans feel the same way," he added. "We intend to do our best to keep these issues before the Congress."

The NICHS and the coalition said scientific evidence is overwhelming that cigarette smoking: causes cancer of the lung, larynx, esophagus, urinary bladder, mouth and pancreas; causes emphysema and chronic bronchitis; is a major risk factor for coronary heart disease; is associated with retarded fetal growth; and is an increased risk for spontaneous abortion and prenatal death.

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COALITION ON SMOKING OR HEALTH

..... A Public Policy Project With
The National Interagency Council on Smoking and Health

The National Interagency Council on Smoking and Health (NICSH) is a voluntary association of health, education, and youth leadership organizations having responsibility or concern with the problem of cigarette smoking on human health. It seeks to provide a cooperative and independent force to inform the public of the harmful effects of cigarette smoking. Thirty (30) national organizations currently comprise the membership of the Council.

Concerned with the emergence of a number of national initiatives in smoking and health, the NICSH Board of Directors in July, 1981 approved a proposal to relocate in Washington, D.C. and focus its resources on public policy issues.

The NICSH, along with many of its member organizations, participated in a National Conference on Smoking OR Health in New York in November, 1981. The 250 conferees in attendance recommended a broad agenda of future actions to advance the cause of smoking prevention in the United States.

The Conference called on the three major voluntary health organizations -- the American Cancer Society, the American Heart Association the the American Lung Association -- to take leadership in establishing a national coalition in Washington, D.C. to implement its priority recommendations in the area of public policy.

These three agencies -- working in close cooperation with the NICSH -- are now in the process of establishing the Coalition on Smoking OR Health to bring these issues to the attention of legislators and federal officials and to promote support for federal smoking prevention education programs.

The NICSH and the Coalition on Smoking OR Health are deeply concerned about the relentless toll in death, debilitation and dollars taken each year by smoking related diseases.

In 1981 the Department of Health and Human Services estimates that the annual health and economic costs of cigarette smoking will total:

- more than 340,000 premature deaths in the United States
- the debilitation of an additional 10 million Americans
- the expenditure of an estimated $41 billion, as follows:
  - -- $13 billion in medical care costs
  - -- $25 billion in lost economic productivity
  - -- $ 3.8 billion in Medicare and Medicaid costs

The NICSH and the Coalition on Smoking OR Health believe that scientific evidence of the health hazards is overwhelming and clearly indicates that cigarette smoking:

- causes cancer of the lung, larnyx, esophagus, urinary bladder, mouth and pancreas
- causes emphysema and chronic bronchitis
- is a major risk factor for coronary heart disease and arteriosclerotic peripheral vascular disease
- is associated with retarded fetal growth, an increased risk for spontaneous abortion and prenatal death

The Coalition on Smoking OR Health will immediately focus its energies on a number of public policy issues, including:

- promoting a substantial increase in the federal excise tax on cigarettes to deter children and youth from starting to smoke
- supporting enactment of the Comprehensive Smoking Prevention Act (H.R. 4957 and S. 1929) which would:
    - -- establish a congressional mandate for the Office of Smoking and Health
    - -- establish a means to coordinate federal smoking and health programs
    - -- strengthen cigarette warning labels
- countering fallacies, myths and misinformation on the health hazards of smoking
- providing a mechanism for coordinating private sector smoking and health programs and establishing an interface with public sector programs
- raising the question of adequate federal protections for Americans against exposure to the toxins in tobacco, which is now exempt from protective controls which govern foods, drugs, substances and products
- questioning the consistency and propriety of national public policies which allocate tax dollars to inform Americans about the health dangers of cigarette smoking and allocate additional tax dollars to sub[illegible]e the production of tobacco.

The Coalition on Smoking OR Health and the member agencies of NICSH stand ready to assist the Congress and the Department of Health and Human Services in providing Americans with the health information they need in order to make an informed choice on whether or not to smoke.

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"Cigarette smoking is the single most preventable cause of death in the United States"

NATIONAL INTERAGENCY COUNCIL ON SMOKING AND HEALTH

Private Sector Member Organizations

American Academy of Pediatrics

American Alliance for Health, Physical Education, Recreation and Dance

American Association of Respiratory Therapy

American Cancer Society

American College of Chest Physicians

American College Health Association

American College of Radiology

American College of Surgeons

American Dental Association

American Heart Association

American Hospital Association

American Lung Association

American Medical Association

American Medical Student Association

American Pharmaceutical Association

American Public Health Association

American School Health Association

Association of State and Territorial Health Officers

Boys Club of America

March of Dimes Birth Defect Foundation

National Board of Young Men's Christian Association

National Board of Young Women's Christian Association

National Congress of Parents and Teachers

National Jogging Association

National League for Nursing

National Medical Association

National Student Nurses Association

Coalition on Smoking OR Health
A PUBLIC POLICY PROJECT WITH THE
NATIONAL INTERAGENCY COUNCIL ON SMOKING AND HEALTH
419 7TH STREET, N.W., WASHINGTON, D.C. 20004

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Chairman, National Interagency
Council on Smoking and Health

March 16, 1982

FORMER FTC COUNSEL TO STAFF
COALITION ON SMOKING OR HEALTH

WASHINGTON, March 16 -- The "Coalition on Smoking OR Health," recently formed to advance the cause of smoking prevention in the United States, has retained Matthew L. Myers, former staff attorney for the Federal Trade Commission as its staff director, it was announced today.

The Washington-based coalition, which is backed by thirty of the nation's leading health, education and youth leadership organizations, will bring smoking prevention issues to the attention to legislators and federal officials and will promote support for federal smoking prevention education programs, according to John D. Kessler, chairman of the coalition's steering committee.

"We are immensely pleased that Mr. Myers has agreed to serve as our staff director" Kessler said. Myers directed the FTC's Staff Report on Cigarette Advertising, released in 1981.

-More-

"CIGARETTE SMOKING IS THE SINGLE MOST PREVENTABLE CAUSE OF DEATH IN THE UNITED STATES"

"The Coalition on Smoking OR Health serves as the public policy action arm of the National Interagency Council on Smoking and Health (NICSH)," Kessler said. NICSH is a voluntary association of leading groups formed in 1967 to inform the public about the dangers of cigarette smoking. The coalition will work closely with three major voluntary health organizations -- the American Cancer Society, the American Heart Association and the American Lung Association.

Testifying today in support of S. 1929 before the Senate Committee on Labor and Human Resources was Charles A. LeMaistre, M.D., President of the University of Texas M.D. Anderson Hospital and Tumor Institute.

"The Coalition on Smoking OR Health represents a banding together of our nation's most prestigious health agencies out of concern for the tragic consequences to Americans of smoking," Dr. LeMaistre said.

The coalition stands ready to work closely with Congress and government at all levels to see that information on the hazards of smoking reaches all segments of the American public, he added.

Robert M. Daugherty, Jr., M.D., Ph.D., chairman of the NICSH said, "through the Coalition on Smoking OR Health we hope to encourage federal initiatives and activities on smoking and health." Dr. Daugherty is dean of the school of medicine at the University of Nevada at Reno.

-More-

"Although the prevalence of smoking is declining," Dr. Daugherty said, "we do not underestimate the difficulty of the task that lies ahead." He added, "The tobacco industry, with its vast resources, is very good at waging campaigns designed to create diversion and doubt in the minds of the public."

"To counter the immense determination of the tobacco industry to keep Americans smoking, our limited resources must be effectively coordinated and the interface with the federal sector maintained," he said. "We hope the coalition will serve to focus our efforts in this critical public health area."

Dr. LeMaistre noted that the lack of information on the health hazards of smoking had contributed to the health and economic costs of cigarette smoking which are estimated at 340,000 premature deaths, health injuries to an estimated 10 million Americans and costs of $41 billion in medical care and economic productivity.

"Can there be any question,"he asked," that cigarette smoking is the single most preventable cause of illness and death in the United States?"

Dr. LeMaistre noted that scientific evidence is overwhelming that cigarette smoking causes cancer of the lung, larynx, esophagus, urinary bladder, mouth and pancreas; causes emphysema and chronic bronchitis; is a major risk factor for coronary heart disease; is associated with retarded fetal growth; and is an increased risk for spontaneous abortion and prenatal death.

Edmund C. Casey, M.D., *President*
Conrad M. Fowler, *President-Elect*
Richard Sinsheimer, *Past-President*
Walter J. Hatcher, *Vice-President*
Edward M. Sewell, M.D., *Vice-President*
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Statement of the American Lung Association
to the House Subcommittee on Health and
the Environment on H.R. 5653, the
Comprehensive Smoking Prevention Education Act

Presented by Stephen Ayres, M.D.

March 5, 1982

I am here to testify in support of H.R. 5653 in my capacity as a volunteer board member of the American Lung Association and Chairman of that organization's Committee on Smoking and Health. In my professional life I am Professor and Chairman of the Department of Medicine, St. Louis University School of Medicine, St. Louis, Missouri.

Although the majority of Americans are aware that smoking is hazardous to health, there is still confusion--even skepticism--about the facts. Much of this confusion is created and perpetrated--deliberately--by the tobacco industry, whose spokespeople constantly tour the country, seeking access to media of all kinds. What they perpetuate is the myth that there is a smoking "controversy" about the health effects of smoking, that the link between cigarette smoking and disease is statistical and therefore suspect, even non-existent.

The facts, of course, are quite otherwise. The link between smoking and disease is one of the most thoroughly documented in medical history. I am introducing into evidence--but will not read now--some of the recent findings from laboratories around the world confirming the relationship between cigarette smoking and human disease. Many different types of studies--30,000 in all and still accumulating--have been performed: epidemiologic studies of death rates; research on effects of stopping smoking; studies of lung function; and examination of tissues at autopsy in smokers and nonsmokers.

In this country alone, 340,000 people die prematurely every year from the effects of cigarette smoking. (1)

At the American Lung Association we are constantly examining the ongoing and new research about the hazards of smoking--on both nonsmokers and smokers. We are alarmed, for example, about radioactive components of cigarette smoke and its effects on smokers themselves and others in their vicinity. Alpha emitters polonium-210 and lead-210 are highly concentrated on tobacco trichomes and insoluble particles in cigarette smoke. The major source of the polonium is phosphate fertilizer, used in growing the tobacco. (2)

Founded in 1904, the American Lung Association includes affiliated associations throughout the U.S., and a medical section, the American Thoracic Society

We are concerned that 75% of the alpha activity of the cigarette smoke may enter the ambient air and is thus available for deposit in the lungs of nearby nonsmokers.

As a volunteer Chairman of the Smoking and Health Committee of the American Lung Association, I am distressed that 60% of Americans polled are unaware that smoking causes most cases of bronchitis and emphysema, for example, that 20% do not know that smoking causes lung cancer, that over 50% do not realize that smoking causes many as opposed to only a few cases of heart attack. (3)

Clearly current federal, state, and private initiatives have been insufficient in conveying the appropriate health messages to important segments of the American public. Hard as they may try, the voluntary agencies alone are fighting an uphill battle with limited resources to fill this informational gap. Some overriding official, national action that would attract constant attention is required. We believe the provisions of H.R. 5653 are important steps to address this crisis.

We favor the provisions on providing statutory standing to the Office on Smoking and Health with the Department of Health and Human Services. There must be a clearly visible focus within the federal government which demonstrates the government's commitment to prevention of disease due to smoking. We endorse the requirement that the Office become more actively involved in the preparation and dissemination of educational and scientific information and we look forward to their actively involving a wide range of federal and private organizations in these activities. At the ALA we rely strongly on the Office on Smoking and Health for fact-finding and documentation.

There is no question that the harm inflicted by cigarettes is more than sufficient to warrant banning production of them. However, in a free society, government--instead of prohibiting production--has at least the responsibility of letting its citizens know the consequences of exposure so they themselves can make informed choices.

Because representatives of a multi-billion dollar industry are so hard at work promoting their self-serving myths and spending an astounding $1 billion each year to promote their lethal product in every media available to them, the American Lung Association believes very strongly that H.R. 5653 is critically important to help inform the public about the real dangers--not the tobacco industry's myths--of smoking.

What concerns us at the American Lung Association is the incredible imbalance between the citizen's exposure to the facts about smoking and the avalanche of cigarette advertising. We find it shocking that cigarettes are the most heavily advertised product in America, that cigarette ads comprise half of all billboard advertising. (4) We are especially concerned that children are not free to make an informed, balanced choice about smoking when they grow up in a society saturated with ads that encourage smoking and present that behavior as something healthy, attractive, sexy. The effects of advertising on children are an overriding issue, in our opinion.

As for rotating and increasing the visibility of warning labels on cigarette packs and in advertising, we think the time has long been ripe for that. We recommend putting the warning message on the face--not the side--of the package. The rotations would alert people constantly about the specific effects of smoking. Obviously, people need more specific information. We also support listing carbon monoxide content as well as tar and nicotine. In addition, we would like to see the additives in cigarettes listed.

In a survey of readers of the American Lung Association's Bulletin a monthly magazine, we found our readers in favor of rotating labels. An especially popular one was: "WARNING: Cigarette smoking is dangerous to health and may cause death from cancer, coronary heart disease, pulmonary emphysema and other diseases." Many favored it because it was the most comprehensive. Some felt it was not strong enough.

It seems clear that Congressional leadership is required to help redress the massive imbalance of cigarette advertising that affects all areas of our society. H.R. 5653 represents the kind of national action desperately needed to begin to counteract the epidemic of smoking-related diseases in this country and to encourage the 53 million smokers to find the way to quit their life-threatening habit. With new self-help and group programs we developed in 1981, at the American Lung Association we are ready to help show those smokers how to quit successfully.

In cooperation with the Office on Smoking and Health, the National Interagency Council on Smoking and Health and the major voluntary organizations such as the American Cancer Society and the American Heart Association, we look forward to increasing our joint efforts to combat this country's number-one preventable public health problem.

The newly organized Coalition on Smoking OR Health provides a unique mechanism for coordinating private sector smoking and health programs and establishing an interface with public sector programs.

Thank you for your attention.

SOURCES

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2. New England Journal of Medicine, 2/11/82

3. Staff Report on the Cigarette Advertising Investigation, FTC, 5/81

4. Staff Report on the Ci[illegible] Advertising Investigation, FTC, 5/81

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THE IMPORTANCE OF THE FEDERAL GOVERNMENT IN THE PREVENTION OF SMOKING-RELATED DISEASES

Testimony in Support of H.R. 5653,
a revised version of H.R. 4957
the Comprehensive Smoking Prevention Education Act by
The American Lung Association

Prepared by Stephen M. Ayres, M.D.
Chairman, Smoking or Health Committee

February 1982

Founded in 1904, the American Lung Association includes affiliated associations throughout the U.S., and a medical section, the American Thoracic Society

may "know" that a particular behavior is harmful even though they do not translate this knowledge into appropriate action. They know but they do not believe. Health education is pitifully primitive in the United States, health educational spots have markedly declined from television programming, and the miniature "warning" on cigarettes and cigarette advertising is ineffective. At one time, when a surgeon general had not been appointed to office, the tobacco industry ridiculed the printed warning by pointing to the non-existence of the individual whose name appeared on the cigarette package.

Rotation of warning labels that would constantly remind potential smokers of specific diseases produced by cigarette smoking each time they reached for a smoke and would have an important effect on converting fact into belief. While clearly less effective than the billion dollar advertising blitz launched annually by the tobacco industry, such rotating labels would be an important first step. A casual inspection of advertising material convinces most observers that its mission is to encourage non-smokers to smoke and to keep smokers smoking. Rotation of labels should be followed by other health educational techniques such as the publication of an anti-smoking message of identical size next to each smoking advertisement. Free choice in a free society is only possible when each individual is informed. Crying "fire" in a crowded theatre is not dissimilar to encouraging one's neighbor to regularly inhale smoke!

SMOKING DOES CAUSE HUMAN DISEASE

Although the tobacco industry characterizes the linkage of cigarette smoking and human illness as the "Smoking and Health Controversy," the only controversy is the unwillingness of that industry to voluntarily phase out cigarette production and to encourage individuals to stop smoking. The evidence establishing the toxic nature of cigarette smoke has been accumulating since the publication of the first report of the Surgeon General in 1964. Since then thousands of articles documenting the harmful effects of cigarette smoking have been published and the United States Public Health Service has issued 13 subsequent reports on smoking and health. In 1976, the National Heart, Lung and Blood Institute, a component of the prestigious National Institutes of Health, concluded its Task Force Report on Prevention, Control and Education in Respiratory Diseases with the following recommendation:

"Cigarette smoking is the single most important risk factor for diseases of the lung. It is known to cause or exacerbate not only the respiratory diseases discussed in this report, but lung cancer, cardiovascular disease and stroke, as well. Reduction, or ideally, elimination of cigarette smoking would have a major impact on national health and on the social and economic costs that are a consequence of smoking-related diseases. The problem of smoking warrants the highest priority in all programs concerned with diseases of the lung. The most important target groups for antismoking programs are preadolescents and adolescents who have not yet started to smoke or in whom the smoking habit is not entrenched."

In a free society, government cannot directly order abolition of destructive behavior but must constantly warn of the consequences of such behavior so that individual citizens can make informed choices. Such destructive behavior accounts for a large component of the annual mortality experience. In 1980, for example, approximately 700,000 died from coronary artery disease, 180,000 from stroke, 105,000 from lung cancer, 30,000 from bladder cancer, 7,500 from esophageal cancer and 60,000 from emphysema and other chronic obstructive pulmonary diseases. The common factor linking these one million deaths, more than half of the total deaths each year, is a demonstrated relationship to cigarette smoking.

Many of these deaths are related to multiple factors including heredity, exposure to environmental agents and excessive dietary cholesterol as well as to cigarette smoking. Cigarette smoking, however, is the single most important factor--many times more important than any other risk factor. Detailed epidemiologic data allow separation of the mortality directly related to smoking.

The Geller-Gesner Tables, published by Robbins and Hall in 1970, have been used by many physicians who practice "prospective medicine" to analyze the life expectancy of individual patients. The table below shows death rates for two hypothetical population samples, a group of 100,000 smokers and another group of 100,000 non-smokers. The number of deaths expected in the subsequent ten years for the two groups of men between the ages of 55 and 59 are shown. Only the four most common causes of death associated with smoking are shown in this calculation.

	Non-Smokers	Smokers
Coronary Artery Disease	6,168	11,434
Lung Cancer	257	3,243
Stroke	1,066	1,600
Emphysema	69	860
Total Deaths Expected	7,560	17,137

This analysis shows that for each 100,000 population sample in the age group 55-59 years, the smoking population will have 9,577 more deaths than the non-smoking population. If one assumes that the total hospital and professional cost of each individual prior to death averaged $10,000, the total excess health cost of the smoking group is close to 100 million dollars. Since there are about five million men in the United States between the ages of 55 and 59, the total excess health cost in that age bracket is five billion dollars.

NEW AND CONFIRMING EVIDENCE IS CONTINUALLY PUBLISHED

Each year a large number of publications from laboratories around the world confirm the relationship between cigarette smoking and human disease. Many different types of studies have been performed--epidemiologic study of death rates, results of stopping smoking, studies of lung function, and examination of tissues at autopsy in smokers and non-smokers. A brief selection of several recent papers follows. Each of these has been published in a highly respected, peer-reviewed medical journal.

Two recent studies have shown increased life expectancy in individuals who discontinued smoking compared to those who continued with the habit. Doll and Peto (British Medical Journal, 2: 1525-1536, 1976) followed 34,000 physicians in Great Britain for 20 years; half of the physicians followed stopped smoking during that period. Deaths from chronic obstructive lung disease decreased by 24% in the physician group while the decline was but 4% in the general public. The death rate from lung cancer in physicians who continued to smoke was 16 times that observed in lifetime non-smokers; the death rate for physicians who had refrained from smoking for from five to nine years was six times that of non-smokers and fell to twice the rate of non-smokers after fifteen years of abstinence. The study also demonstrated an important relationship between the duration and the amount of smoking and the occurrence of lung cancer, dose-response relationships that strongly support the concept of direct causality.

Friedman and associates (New England Journal of Medicine 300, 213-217, 1979) followed 4004 men and women over an eleven year period after an initial evaluation. The mortality rate in smokers was 2.6 greater than that in non-smokers and the differences could not be explained by any of 48 baseline characteristics. In a later study, the same group (New England Journal of Medicine 304 1407-1410, 1981) studied the effects of stopping cigarette smoking on death rates. The overall death rate was 2.22 greater in those who did not stop smoking compared to those who did. Of great importance was the observation that among those individuals who had coronary artery disease at the time of the initial observation, the death rate in those who continued to smoke was 3.92 times that in the group that stopped smoking.

Evidence that cigarette smoking may produce an acute myocardial infarction (heart attack) was presented by Hertz et al (Journal of the American Medical Association 246 851-853, 1981). Heart attacks were 2.27 times more common in the heavy smokers aged 35-49 compared to non-smokers; they were 1.95 times more common in the heavy smokers aged 35-65.

Both lung function studies and pathologic studies on human lungs have shown the deleterious effects of cigarette smoking. White and Froeb (New England Journal of Medicine 302 720-723, 1980) observed that lung function was 31% lower in individuals smoking more than two packs of cigarettes each day compared to non-smokers. Of great interest, lung function was 13% lower in non-smokers working in a smoke-filled environment compared to those in a fresh atmosphere. Cosio et al

The environmental sanitation movement of the early Twentieth Century with an emphasis on pure drinking water and sanitary waste disposal provided clear evidence that maintenance of the public health was a major responsibility of government. More recently, the widespread adoption of cigarette smoking has led to a new epidemic of preventable deaths that can only be eliminated by direct action of public health authorities. Like the Black Death or Plague of 1348-1350 which killed one third of the population, cigarette smoking leads to the premature deaths of large numbers of a nation's population. Adoption of H.R. 5653, the "Comprehensive Smoking Prevention Act" would place the United States government squarely in the role of defender of the public health.

The Federal Government must attempt to eliminate as many of the hundreds of thousands of deaths due to cigarette smoking as possible and to reduce the billions of dollars spent for the care of people with tobacco-related diseases. Every effort must be made to discourage young people from starting smoking and to help confirmed smokers stop smoking. The increase in cigarette smoking among adolescent girls has been particularly alarming and there is reason to believe that a group of societal pressures are responsible for this situation. Lung cancer was at one time extremely rare in women; its dramatic increase in the past decade is a stark reminder of the risks of adolescent cigarette smoking.

KNOWLEDGE AND BELIEF

Why do people smoke? The Roper Report commissioned by the tobacco industry warned that most people knew that cigarette smoking was harmful and that many smokers desired to stop. This public opinion organization detailed a series of problems that threatened the continued viability of the tobacco industry and emphasized particularly the spread of health knowledge among the public and the growing activism of non-smokers.

Unfortunately, the knowledge that cigarette smoking is dangerous is not necessarily translated into abstinence. Behavioral scientists have developed the "health belief" model to explain why individuals smokers had significantly more evidence of inflammation in the smallest areas of the airways and that this inflammation was probably reversible when smoking was discontinued. Hale et al in an article published in the same journal demonstrated for the first time that smokers dying without obvious heart disease had thickening of the pulmonary blood vessels. These data suggest a response to reduced oxygen concentrations in the smaller airways related to cigarette smoking.

The toxicity of cigarette smoke to innocent bystanders as well as to smokers was shown by a recent study of Dahms et al. (Chest 80 530-534, 1981). Ten patients with bronchial asthma and ten normal individuals were exposed to cigarette smoke in an environmental chamber. Pulmonary function tests (the $FEV_{1.0}$) decreased 21% in the asthmatics but not in the normals. The asthmatic patients were not particularly sick and were ambulatory. A substantial percentage of the general population has the sort of reactive airways demonstrated by the asthmatic so that the study emphasizes a major public health concern.

TESTIMONY
OF
JOHN A. OATES, M.D.

CHAIRMAN
SUBCOMMITTEE ON SMOKING
AMERICAN HEART ASSOCIATION

Submitted to the

COMMITTEE ON LABOR AND HUMAN RESOURCES
U.S. SENATE

MARCH 16, 1982

Mr. Chairman and members of the Committee on Labor and Human Resources, my name is John A. Oates, M.D. I am Professor of Medicine and Pharmacology at Vanderbilt University and Chairman of the Subcommittee on Smoking of the American Heart Association.

The American Heart Association enthusiastically supports S. 1929 the "Comprehensive Smoking Act of 1981." As you may know, the American Heart Association is a nonprofit voluntary health organization with over 119,000 members and almost 2 million other volunteers who are dedicated to the reduction of premature death and disability from cardiovascular diseases.[1]

Cardiovascular diseases kill nearly one million Americans each year. This is more than all other causes combined. Heart attacks, the nation's number one killer, claims most of these lives. This year, as many as 1.5 million Americans can be expected to have a heart attack and about 550,000 of them will die. The survivors will join over 4 million Americans who have a history of coronary disease.[2]

These figures have special significance because cigarette smoking has been firmly implicated as a major contributor to the occurrence of heart attacks, sudden death, peripheral vascular disease and it greatly aggravates other forms of cardiovascular diseases. Cigarette smokers are more likely, than non-smokers, to suffer a heart attack, more likely to die from these attacks and more likely to die suddenly. This effect is directly related to the amount smoked with heavy smokers being at three times the risk of non-smokers. Fortunately, ceasing to smoke reduces the risk toward that of nonsmokers.[3]

Based on data from the Framingham Heart Study[4], we estimate that over 174,000 Americans will develop peripheral vascular disease this year. While, twenty percent of these people will be diabetics, 70% of the remainder will be cigarette smokers.[5] Moreover, diabetics who also smoke are at even greater risk.[6] The link of cigarette smoking to peripheral vascular disease is consistent and independent of diabetes and other risk factors and related directly to the number of cigarettes smoked.[7]

Perhaps most significant is the fact that when people stop smoking, their risk of these cardiovascular diseases gradually returns to normal. The risk of fatal and non fatal heart attacks among ex-smokers is similar to non-smokers in about 10 years.[8-9] The risk of peripheral vascular disease is similarly reduced to that of a non-smoker in about 5 years.[10]

The evidence incriminating cigarette smoking as a major risk factor for heart attack and peripheral vascular disease was judged by an expert panel of the American Heart Association to be conclusive.[11] They further concluded that "Theoretically, cigarette smoking is the most preventable cause of these cardiovascular diseases and mortality therefrom".[3]

In spite of the overwhelming evidence linking cigarette smoking to cardiovascular and other diseases, over 52 million Americans are still smoking.[12] However, two thirds of these smokers would like to quit and most have tried to do so.[12] Following an unsuccessful attempt to quit, many of these smokers tend to switch to low tar and nicotine cigarettes.[13-14] This is evidenced by the continuing rise in the market share of these cigarettes to 60.9% of all sales in 1981.[15]

This development is alarming because the evidence[13] suggests that many people switch to low tar and nicotine in an effort to lower their risk of adverse health effect. While switching to these cigarettes may lower the risk for some diseases, there is no evidence of a reduction in risk for cardiovascular diseases.[14] In fact, recent evidence from the Framingham Heart Study[16], suggests that low tar and nicotine cigarettes may even increase the risk of cardiovascular disease. This could be a very dangerous development since most cigarette-related deaths are from heart attacks.[9]

The American Heart Association is committed to helping smokers who want to quit and preventing children from starting to smoke. Accordingly, we wholeheartedly support the passage of S. 1929, the "Comprehensive Smoking Prevention Education Act of 1981". The provision of a congressionally mandated smoking and health program is very important. The proposed role of the federal smoking and health program will be instrumental in combining and coordinating the efforts of the public and private sectors to address the problem of cigarette smoking. This is a clear case where a relatively small federal effort can be used to mobilize enormous private sector resources to address a major health problem.

The provision requiring the rotation of five new warning statements on cigarette packages and advertisements is needed to better inform the public of the specific dangers of cigarette smoking. The rationale for the requirement of a warning statement in the first place, was to inform consumers of the health hazard. However, the FTC[17] has recently concluded that the current warning statement, which has been used on packages and advertisement since 1972, is overexposed and worn out. This conclusion was supported in part by a study by Starch Message Report Service which found that only 2.4% of adults exposed to cigarette ads reads the Surgeon General's warnings. This should come as no surprise since any message presented exactly the same way will soon become so familiar that it will lose its effectiveness.[18-19] Furthermore, since cigarette companies vary the copy of their advertisements to avoid the "wear out" effect, it seems reasonable to conclude that the warning statement requires no less.

Brodsky and Myrowitz,[20] in a study of cigarette billboards, found the warning statement to be written uniformly on all billboards and in a manner similar to that used in newspaper and magazines. Individual letters in the warning consistently appeared the same, i.e., reduced detail. When compared with the detail and size of the letters in the brand name, the two largest observed warning statements were 38 and 17 times smaller. These two optometrists concluded from their test that while the brand name was visible, the warning was not.

This amply supports the need for varied warning statements in a format that is visible. However, the Tobacco Institute[21] in response to the FTC Staff Report, argued that the effectiveness of the cigarette advertisement and warning statement must be judged by different criteria. The rationale is that "the former (advertisement) must achieve consumer recall for a particular brand among numerous other brands, all of which present competing and conflicting messages while the latter (warning) appears consistently to the consumer in every advertisement and on every pack of cigarettes." This dual criteria is not only unsupported but unsupportable. If the warning statement has been "remarkably effective" by being presented consistently the same way then it would seem that brand recall would be enhanced if the portrayed image was maintained unchanged over the years. For example, the Marlboro advertisement should be most effective if the original ad copy was continued. Yet, the copy is changed and changed frequently. Different cowboys are used in different settings. Additionally, Brown and Williamson made several changes in its very successful Barclay campaign last year.

The Tobacco Institute also noted that cigarette advertising uses a multitude of images to appeal to different markets. This is indeed consistent with the concept of market segmentation. The logical extension of this is that the warning must also be tailored to appeal to differing consumers. For example, smokers with a history of heart disease in their families may find a warning on smoking and heart attack much more personally relevant than a general message.

Moreover, the argument that cigarette advertising is limited to the print media while consumers are exposed in all media to information on the hazards of smoking is misleading. The number of anti-smoking PSA aired on television since the ban on cigarette commercials has been drastically reduced with hardly any being shown during prime time. Additionally, information on smoking and health in the print media has been equally scarce. It has even been suggested by some[22-23] that the tobacco industry may be using its advertising as a leverage to discourage coverage on the hazards of smoking in the print media. For whatever reason, the broadcast and print media is largely inaccessible for informing the public of the dangers of smoking.

Meanwhile, the so called brand advertising goes on at the tune of one billion dollars per year.[17] Regardless of the intent, non-smokers and children are heavily exposed to this advertising. It would be very difficult to argue convincingly that some non-smokers, especially children and teenagers, are not affected by this constant portrayal of smokers as being young, attractive, healthy and enjoying an adventurous and pleasurable lifestyle. With this kind of bombardment with no effective counter, it is not hard to understand why 53% of smokers do not know or believe that smoking causes many cases of heart attack.[24]

For the above reasons, the warning label becomes a very important medium to get more information to the public. The American Heart Association has made and will continue to make efforts to better inform the public about hazards of smoking. However, we do not believe that we have adequate access to the media to fully inform the public. Our efforts will have a greater chance of success if the clearly deceptive intention of cigarette advertising is attenuated by including an effective warning. The warning is yet another way, at no cost, in which the federal government can effectively assist the private sector in educating the public about the hazard of cigarette smoking.

Therefore, the American Heart Association strongly supports the adoption of the five specific warnings proposed in S. 1929. We further propose the incorporation of the "circle and arrow" format, recommended by the FTC[17], to display the warning on advertisements. This format would significantly enhance noticeability of the warning. Finally, requiring that the warning letters measure at least 25% of the maximum brand name letters and be of the same proportionate detail would much improve its visibility.[18] These simple measures, at no cost, would contribute greatly to the education efforts of the private sector.

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TESTIMONY
OF
ROBERT M. DAUGHERTY, JR., M.D., PH.D.

CHAIRMAN
NATIONAL INTERAGENCY COUNCIL
ON
SMOKING AND HEALTH

SUBMITTED TO THE
COMMITTEE ON LABOR AND HUMAN RESOURCES
U.S. SENATE

MARCH 16, 1982

Mr. Chairman, Members of the Committee on Labor and Human Resources, I am Robert Daugherty, Jr., M.D., Ph.D., Dean of the School of Medicine, University of Nevada and the present Chairman of National Interagency Council on Smoking and Health (NICSH).

The National Interagency Council on Smoking and Health is a voluntary association of health, education and youth leadership organizations having responsibility or concern with the problem of cigarette smoking on human health. It seeks to provide a cooperative and independent force to inform the public of the harmful effects of cigarette smoking. Thirty national organizations currently comprise the membership of the Council. A list of those organizations is attached to my statement.

The NICSH, through its member organizations and 80 local interagency councils, has and will continue its efforts to educate people about the dangers of cigarette smoking and assist these smokers who want to quit. The technical information and other service we receive from the federal sector facilitate the more efficient utilization of our limited resources. On the other hand, the Office on Smoking and Health, with its limited budget, must rely heavily on the prviate sector to carry the message to the public. This federal-private interface is largely responsible for the continuing downward trend in smoking among Americans.

While the prevalence of smoking is declining[1-2] we do not underestimate the difficulty of the task that lies ahead. The tobacco industry, with its vast resources is very good at waging campaigns designed to create diversion and doubt in the minds of the public. Since the 1978 public opinion survey conducted for the Tobacco Institute by the Roper Organization[3] recommended these and other tactics, such campaigns have proliferated in the media. Last month, the Tobacco Institute launched what Peter Sparker, Vice-President Public Information,[4] called "the biggest campaign we've ever done". The campaign will include nine insertions in national editions of Newsweek, People, Sports Illustrated, Time, TV Guide, and U.S. News and World Report during 1982. The cost of a campaign of this magnitude is estimated at well in excess of $1,000,000. Considering the conclusive evidence linking cigarette smoking to disease, a campaign with the theme "Weigh Both Sides Before You Take Sides" can only be designed to create confusion.

To counter the immense determination of the tobacco industry to keep Americans smoking, our limited resources must be effectively coordinated and the interface with the federal sector maintained. The NICSH is determined to play its role as coordinator in the private sector. However, the commitment of the federal government to perform its role has become a source of great concern. As you know, the Office of Management and Budget has twice tried to "line out" the Office on Smoking and Health (OSH). Fortunately, DHHS Secretary Schweiker has been able to get the funding restored. The $2-3 million budget of OSH is miniscule when compared to over $1 billion[5] used to promote the sale of cigarettes. Yet, it provides an essential function to the private organization that must get the job done.

S. 1929 would assure that OSH continue to provide this vital function. A secure existence for OSH with delineated functions and objectives, would also be a significant stimulant to the private sector.

The establishment of an Interagency Committee within OSH to coordinate all federal activities that relate to smoking is a very important provision of S. 1929. By requiring federal agencies to coordinate, as the private agencies now do, will further enhance the quality and efficiency of federal performance. Considering the dim prospect for increasing federal outlays in this area, efficiency is critical.

The provision of S. 1929 requiring the rotation of five new warning statements on cigarette packages and advertisements is immensely important. The FTC[5] has shown convincingly that the current warning is "worn out". Immediate action is needed to make this an effective medium to reach the public with information on the hazard of smoking. This requirement is something which the federal government can do, at no cost, to supplement private efforts. Its importance cannot be overemphasized since the broadcast[6] and print[7-8] are largely inaccessible for communicating information on the effects of smoking.

The Tobacco Institute[9] has referred to the rotational warning as a "cumbersome system", implying a burden to the industry. However, an observance of the copy of cigarette advertisements point out how ridiculous this claim is. Brown and Williamson's Barclay ad campaign used at least seven completely different or variations in the copy. This was rated one of the "top ten" campaigns for 1981.[10] In accordance with this provision in S. 1929, the same warning statement would have been used on all these variations. The point is that cigarette ad copy is changed and changed frequently to avoid "wear out". Requiring a label change once a year is indeed a simple matter.

The Tobacco Institute has also argued that this represents increased regulation on an over-regulated industry. This argument also lacks merit. It overlooks the fact that tobacco and tobacco products are either exempt by agency determination or specifically by statute from those laws which were enacted by the Congress and in particular this Subcommittee to protect the health and well being of the American consumer. The Food and Drug Administration has by agency determination ruled that tobacco and tobacco products are neither 'foods' nor 'drugs' to be regulated under the Food, Drug and Cosmetic Act. The Consumer Product Safety Act specifically exempts by statute tobacco and tobacco products from being defined as a "product" to be regulated under that Act. The Federal [illegible] Substances Act specifically exempts tobacco and tobacco products [illegible] being considered a "hazardous product" under that Act. Tobacco and tobacco products are also not regulated under the Toxic Substances Act.

So, although the Congress of the United States has declared cigarettes to be dangerous to ones health, tobacco and tobacco products have escaped being regulated under those Acts which were enacted by the Congress to specifically protect consumers from health and safety risks. While tobacco and tobacco products cannot be considered to be a food under the Food, Drug and Cosmetic Act, I find it somewhat ironic that it continues to be exported as a food under the Food for Peace program.

The argument that the provision of more disease specific warning labels is "excessively regulatory" and "paternalistic" is ludicrous. To the contrary, this legislation represents a minimum of government regulation especially when considered against the exception to the federal laws I have just noted.

S. 1929 would give the consumer the necessary information on the specific health hazards of cigarette smoking so that the consumer would be able to make an informed choice as to whether or not he or she smoked. No one is being denied the right to smoke under this legislation nor are any restraints on sales or distribution being imposed on the cigarette manufacturing industry. This legislation seeks only to provide consumers the right to know all of the health hazards of cigarette smoking.

The National Interagency Council on Smoking and Health stand ready to do their parts in working closely with the Congress and the Federal government in a stronger partnership to ensure that information on the hazards of smoking is reaching Americans everywhere.

Thank you.

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TESTIMONY
On Behalf
of
THE AMERICAN CANCER SOCIETY
Before
The Committee on Labor and Human Resources
U.S. Senate

March 5, 1982

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The American Cancer Society, a voluntary health organization with over 2 million active volunteers dedicated to fighting cancer, is privileged to submit Testimony today. Because of the mandate of the American Cancer Society it is most fitting that we offer our testimony in strong support of S. 1929, the Comprehensive Smoking Prevention Education Act of 1981.

We would like to commend you, Mr. Chairman, for your leadership and your foresight in sponsoring this important preventive health care measure. We are pleased to present testimony as part of the new Smoking or Health Coalition. The work that the American Cancer Society will be doing on this bill together with the American Heart Association, the American Lung Association and the National Interagency Council on Smoking and Health is just the beginning of the work of a coalition dedicated to educating Americans to the dangers of smoking. We are all extremely excited about the potential impact on the smoking problem in this country resulting from the joining of forces of our organizations. The reason we are presenting testimony together is to underscore the amount of energy that our organizations are willing to exert to encourage this vital health effort.

The reasons for our dedication are clear. Today, 54 million Americans will light up a cigarette and we know that smoking will contribute to the death of over 300,000 of them this year alone. 430,000 Americans will die from all forms of cancer in 19[illegible]. According to the February 22 Surgeon General's report on the Health Consequences of Smoking 129,000 of those deaths will be caused by the use of tobacco products.[1]

85% of the 111,000 lung cancer deaths this year will be smoking related. We know from the Surgeon General's report that the overall cancer death rates of male smokers are approximately double those of nonsmokers and for female smokers the death rate is approximately 30 percent higher.

The Surgeon General found that cigarette smoking was a major cause of lung, laryngeal, oral cavity, and esophageal cancer. It was also found to be a contributory factor to bladder, kidney and pancreatic cancer. In addition, he noted that epidemiological studies suggest an association between cigarette smoking and stomach cancer to a possible association between smoking and uterine and cervical cancer.

What is even worse, those cancers most closely associated with cigarette smoking (lung, esophageal, laryngeal, oral cavity and pancreatic cancer), can be the most difficult ones to treat, the cancers with the least hope for survival. For example, the overall 5-year survival rate for lung cancer is only 10%; for cancer of esophagus, 4% and for cancer of the pancreas, 2%.[2]

When the Surgeon General released his report on February 22 of this year, the President of the American Cancer Society, Dr. Robert Hutter, said:

> "When we hear of a bad accident involving 20 or 30 people everyone is horrified and officials start clamoring for more consumer protection. Here we have a situation in which two to three hundred people a day are being killed by tobacco-related cancers and these people have been given virtually no protection at all."

As an organization working with the victims of cigarette smoking, we can state with conviction to the members of this committee that dying from lung cancer is one of the most awful experiences a human being can go through. To permit people to kill themselves this way without making the hazards amply clear is an irresponsible act that ACS, as a voluntary health agency, and you, as legislators, must not allow to continue.

The American Cancer Society urges the sponsors of this legislation to maintain a formal Office of Smoking and Health. Such an office dedicated solely to educating Americans to the dangers of smoking and, in addition, working to eliminate the American smoking habit is vital to the voluntary health sector in real terms as well as symbolically. We need a group of knowledgable professionals at the federal level in such an office committed to this cause.

We are also pleased that the Chairman has shown an interest in adding to his legislation an amendment which would require that all cigarette companies list with the Secretary of HHS all tobacco additives. We are not asking that the trade secrets in the tobacco industry be revealed. However, the ACS questions why the cigarette industry has, for so long been exempt from any requirement to list their additives when most other consumable consumer goods have not had such an exemption.

In addition, we are well aware that cigarette smoking is dangerous. It is vital, that at the very least, government scientists be given an opportunity to test the health impacts of burning and inhaling such flavoring additives as cocoa husks. The scientific and medical communities must also be allowed to test these additives so as to protect the 54 million smokers and for those other millions whom we are not reaching through education efforts who may yet start to smoke.

I know the labeling provisions of the legislation raise some questions. I cannot say for sure that six rotating labels listing exactly the ill effects of cigarette smoking will make the difference. I can say that in 1979 13.5% of boys aged 15 and 16 and 11.8% of girls that age are regular smokers. I can say that at ages 17 and 18, the incidence increases to 19.3% for boys and 26.2% for girls. Also, the percentage of girls age 17 and 18 who smoke has risen sharply since 1974. One further point, by 1983, according to statistics from the National Cancer Institute, lung cancer will outdistance breast cancer as the number one cancer killer of women.[3]

Our children are risking their lives, possibly because of clever advertising, combined with the various reasons that influenced so many people to begin smoking years ago: peer pressure, peer imitation, saying it looks so sophisticated, grown-up, etc. At the very least, we must give them the best information about the hazards of cigarettes to permit them to make an informed decision. An FTC staff study came to the following conclusion:

> "...many [smokers] are unaware of the existence of the relationship between smoking and some of its most serious and widespread health consequences, such as heart disease...Some of the

> health consequences of smoking, such as lung cancer and emphysema, are more well known. However, even for lung cancer, the most well known health effect, some substantial gaps in consumer knowledge are evident."[4]

We have an obligation to fill those gaps, especially for the young. How many 17 year old girls who are lighting up a cigarette today comprehend that they could get oral cavity cancer and would have to undergo the difficult and painful rehabilitation that is necessary to overcome the disabilities associated with that disease. Not enough!

Mr. Chairman, the Congress's own research agency, the Office of Technology Assessment, in its ASSESSMENT OF TECHNOLOGIES FOR DETERMINING CANCER RISKS FROM THE ENVIRONMENT, dated June 1981, states that tobacco smoking "is the single most important preventable environmental factor contributing to illness, disability, and death in the United States." The OTA report sites a WHO reference stating "smoking-related diseases are such important causes of disability and premature death in developing countries that the control of cigarette smoking could do more to improve health and prolong life in these countries than any single action in the whole field of preventive medicine."

Further, the OTA report adds, "tobacco is know to contribute more heavily to the number of cancer deaths than any other single substance."

Members of this Committee, we urge you and your colleagues in both Houses of the Congress to pass this measure into public law as quickly as your considerations and processes will allow. The American Cancer Society believes this bill can make a difference to the health of our country. We look to you to help us in this crusade!

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2/ 1982 Cancer Facts & Figures, American Cancer Society, Inc., p. 19.

3/ Dangers of Smoking, American Cancer Society, Inc., 1980, p. 57 & 66-68.

4/ Staff Report on the Cigarette Advertising Investigation, Federal Trade Commission, May, 1981, p. 3-45.

The **Chairman**. Thank you, Dr. LeMaistre. We appreciate having you with us today, and we appreciate the great work that you people at the M. D. Anderson Clinic have really done through the years in so many areas of cancer.

Dr. **LeMaistre**. Thank you, sir.

The **Chairman**. I have been through your facility, and I am very, very impressed with it.

Dr. **LeMaistre**. We were very proud to have you there, sir.

The **Chairman**. Thank you.

You have said that the newly founded Coalition on Smoking or Health represents 30 different health education and youth leadership groups. Why have they now banded together in this smoking prevention education effort?

Dr. **LeMaistre**. Mr. Chairman, I think those of us who have been involved in the question of smoking and its effect upon the health of the American public have wondered that for many years.

A conference was held this past fall in New York, sponsored originally by the American Cancer Society and joined by the American Lung and American Heart Associations, along with at that time 21 other agencies. They considered the problem of smoking and they found much in common that they could do collectively that previously they had been attempting separately. All of these agencies have been independently concerned about this problem for a very long time.

However, the new coalition, working through the National Interagency Council on Smoking and Health, is thought to be a far more effective and efficient way of bringing the feelings of all of the volunteers that these agencies represent to the attention of those who might be interested in hearing their views.

The **Chairman**. You have provided testimony representing perhaps the last coalition of interested health groups on this subject, and I am pleased to see the high degree of interest and cooperation directed at what I personally consider to be an important problem.

However, with such tremendous effort being generated outside of the Federal Government, within the private sector, why do you think this legislation is necessary?

Dr. **LeMaistre**. Mr. Chairman, the legislation requiring disclosure of information and new labeling is important for several reasons. I consider this bill an essential first step toward other bills that will lead to the education of the American public; in particular, the younger American public that has not yet made the decision to smoke, so that they may have the freedom of an informed choice.

In particular, the information that is becoming increasingly alarming about additives that you just heard Dr. John Holbrook testify to is a major concern of all of these organizations. This is relatively new information, as the health consequences of cigarettes have changed over the years as the cigarette also has changed.

You have also heard this morning in testimony that, whereas the majority of the American public does generally appreciate the hazards of cigarette smoking, they are not well informed on the specific hazards, and the relationship to lung cancer is better known than the relationship to heart disease and perhaps even to emphysema.

So, for all of these reasons, it is felt that the only mechanism by which full information can be obtained is by action of Congress at this point in time. As I pointed out early on, there have been repeated reports by the Surgeon General, 30,000 scientific reports, all of which have given the opportunity for a response on the basis of scientific evidence, whether epidemiologic or otherwise. That has not been forthcoming. The American public is not reassured that they know all that they need to know about the hazards of cigarette smoking.

Those of us who work in the field see that the problem is aggravated by the fact that young people are still taking up the cigarette smoking habit, an alarming habit. For that specific reason, if for no other—but there are many other reasons—I feel these agencies now must band together to support any action, particularly such intelligent and far-reaching action as contained in this bill.

The CHAIRMAN. You stated in your testimony, "Can there be any question about the stark fact that cigarette smoking is the single most preventable cause of illness and death in the United States?" Yet apparently some scientists do not accept the thousands of studies—you indicated 30,000 studies—linking cigarette smoking to illness. Now, how do you account for their skepticism?

Dr. LEMAISTRE. I have never been able to account for their skepticism in this. I was one of those skeptics before I served on the Surgeon General's advisory committee that reported in 1964 on the consequences of cigarette smoking. I was a pipe smoker and a cigar smoker. Since I have become intimately acquainted with the evidence, I must say my position has changed.

On the other hand, I think that the mounting evidence is narrowing the number—though they still remain vocal—of scientists who have distinguished records in terms of judgment on such issues as this and the social consequences of smoking. I could not attempt to explain their position, Senator, except to say that our belief is that they do wish to have more information. I believe that the disclosure of these additives and the research that will follow on defining their hazards might provide them with that kind of information that would be more conclusive on one side or the other.

I am not here to plead for anything more than a disclosure of information, factual information, that clearly is not coming through in either advertising or on cigarette packages today.

The CHAIRMAN. Do you think that if we had these warning labels that we have provided in this bill it will have a significant reduction in those who smoke?

Dr. LEMAISTRE. Senator Hatch, I particularly feel that rotating the warnings and focusing upon specific issues of heart disease or of lung disease or of prenatal consequences for the pregnant mother, will have direct consequences for individuals who might be electing to smoke, especially if the individual is pregnant or has already had some warning of heart or lung disease or some family history of cancer.

The impact of that is far greater than the general cigarette package message, which has worn thin over the years. So yes, I do believe it will have a specific impact and aid in an intelligent decision by those who choose to read it.

The CHAIRMAN. In your opinion, how would scientific research be aided by having tobacco companies provide information regarding the chemicals used in the manufacture of cigarettes? Now, you have indicated that that would be very helpful, and we have had Dr. Holbrook's testimony.

Dr. LEMAISTRE. In the bill before the Senate, the disclosure, on a confidential basis in order to protect trade secrets, would allow at least the certain knowledge of what is being added. Those substances then could be the subject of research to find out, as Dr. Holbrook so well stated, the consequences of burning them at between 800 and 1,000 degrees—the pyrolysis that changes those products to determine whether they are indeed harmful or not and whether the harm comes from the fact that their consequences effect the lungs, or heart, or contribute to cancer.

Indeed, that might encourage the tobacco companies to find safer additives.

The CHAIRMAN. In your testimony, you indicated what you described as special treatment of tobacco companies and products. How do you account for that? What do you mean by that?

Dr. LEMAISTRE. I have no knowledge, as Senator East mentioned earlier, of the political complexities that overwhelm many of us, and I agree with him. Sometimes they are far more complex, with greater variables than we see in the scientific world. I stated my observations on the basis of history. I did not try to attribute cause or effect to that. I do think the record is clear, though, Senator.

The CHAIRMAN. Thank you.

Senator East?

Senator EAST. Thank you, Mr. Chairman.

Doctor, I wish to again thank you for coming and helping us in this testimony and hearing.

As you have been here this morning, I have pursued this theme to some extent with the other people from the medical community and from the perspective of medical science, concentrating on the word science here.

You used a phrase which we hear a great deal of in this effort to build a base of support for this legislation. The first finding of this bill, under section 2, says that Congress finds that "cigarette smoking is the largest preventable cause of illness and premature death in the United States," and you used that phrase, "the largest preventable cause of illness."

I am just curious. On this matter of scientific causation, and looking at it in a way that people in the scientific community would want to look at it, not to have it politicized but to have it be meaningful, how could one say with total assurance and as a scientific finding that it is the largest preventable cause of illness?

For example, I could see how, conceivably, you might have facts. I am not saying they are there, but one might be able to establish, as a matter of scientific causation, that a particular thing is the largest cause of illness and premature death, because raw statistics might conceivably bear it out in some way—driving, alcohol, salt, sugar, obesity, nutrition, and so on and so forth—that we might be able to establish that.

But when we say preventable, for example, just throwing in that adjective, which greatly ups the scientific ante here, how do we know, as a matter of science, that it is easier to prevent smoking than other relative health problems? How could we say that with assurance? First I hear scientists in here saying it is addictive and it is very difficult to give up; then I hear scientists coming in and saying, "Oh, it is the most preventable"; suggesting it is easy and you just stop doing it.

Now, I am not saying who is right or who is wrong. What I am saying is, it strikes me that when you move into an area of this kind, and science seems to be butting heads, and words are being used rather casually, getting back to my fundamental concern about scientific causation, whether, the scientific community and the medical science community here have not been unwittingly pulled into a great political movement of some kind that has great political currency with some individuals and groups in parts of the country, getting back to the need now to maintain the integrity of science, I query whether we are not getting a little casual here with wording. "Preventable": are there scientific studies showing that it is easier to give up smoking than to, say, throw away the saltshaker or to give up sugar or to do this or that or the other thing or to correct driving practices? I am just curious. I am not saying what my conclusion would be. I am just troubled with scientists coming in and proclaiming findings, when in fact, in their scientific heart of hearts, they would say, "Oh, well, we don't really quite mean that"; that's politics.

I think science has a separate responsibility here, and that is to maintain its integrity, maintain its commitment to that which is demonstrable and provable with data and quantification. When I see adjectives sprinkled through and I see pieces of legislation here with grandiose conclusions of that kind, I wonder if there is the scientific evidence to back it up or whether it is not just casual conclusions that further a cause.

What would be your response? This word "preventable" I do find intriguing.

Dr. **LEMAISTRE.** Senator East, I feel that everyone who has attempted to respond to that question has acknowledged the credibility of your position from the standpoint of the quantitative aspects of whether it is easier to prevent this or that. Clearly, it is not an easy task to prevent cigarette smoking. We have been trying, to my certain knowledge and involvement in this, since 1962, which I first became interested in this question.

The original Surgeon General's Committee that reported in 1964 experienced the same kind of thought process that you are going through now. If you will go back and look at the committee's conclusions, the strong causal relationships were drawn to reflect the evidence available at that time. Therefore, although the evidence was highly suspicious for cardiovascular disease and highly suspicious for pulmonary disease, we focused of the causality in the relationship with lung cancer, primarily because that is where the evidence really allowed us to go at that time.

Progressively, over the years, other evidence has been added. Indeed, because of the publication of that report, in 1964, 18 million Americans who had been smoking no longer smoked. They were carefully followed as cohorts of those who continued to smoke, and that today still provides the best evidence that stopping smoking decreases the risk of heart attack. So there are factual examples by which one can bolster each of the scientific judgments based on epidemiologic data. These data include both retrospective data, which I consider exceedingly sound, from Great Britain and this country and prospective data in which groups have been studied. Both enhance the integrity of the scientific conclusions.

Coming to your specific point, until we have comprehensive health education in our schools of high quality, from grades K through 12, in my opinion, that allows young people to be fully acquainted with the known facts, there will not be an answer to your question about how preventable this really is in terms of a practical application. But, theoretically, it should be preventable.

The tragic consequences extend far beyond disease. In my hometown, we had a very tragic fire involving young people with whom I am closely associated only a few weeks ago in a hotel, caused by a cigarette. Over one-half of the hospital fires, a place where I work every day, and over one-half of the fatal home fires are caused by cigarettes. That evidence is irrefutable in terms of why the causes of fires are usually determined.

So these factors come together for something that is not a way of life that should be acceptable to the American youngster. I do believe, as a teacher, that young people can be taught about the problems associated with cigarettes in terms of a fair representation of the truth. I have sufficient respect for the young people of this country to let them make their own decisions about which risks of being ill and of dying they are willing to accept. I defend their right to choose those risks, just as I do. I jaywalked coming over here today, and that was an unnecessary risk. But, by the same token, I think that it is not a question of just pure empiricism on prevention. We are now preventing some young people from taking up the habit through better information every year.

Senator **East**. But, you see, in the legislation here, it says, "The Congress finds that," meaning a scientific finding, "it is the largest preventable," and you are suggesting that, theoretically, that sort of a supposition really is not scientific fact.

For example, let's say we determine in a real scientific study that it is not easy to prevent smoking; therefore, it might direct a public policy—I am just hypothetically speaking—whereby the Government would acknowledge, well, we cannot get people to stop smoking; I'll tell you what let's do. Let's try to develop a safer cigarette, you see, because it isn't preventable in the sense it is difficult to get people to stop.

OTHER THINGS

It may be easier to get people to change their behavioral pattern—again, the ones I have worked over this morning, salt, sugar, nutrition, exercise, the whole phalanx of things that go together to make a composite for good health practice. And so when we are told, well, this is easy to do, it is preventable, I don't know that it is any more preventable than these other things, and if scientific findings show that it is not, why not direct our talents and our efforts, rather than in a direct prohibition, which I submit this bill does, why not direct it—we could still inform people of the relative health problems of this or any other thing—why not devote research to perhaps finding a safe cigarette? Then we would be home free. Then we would really have solved the health problem coming out of smoking.

But what I find here is somewhat of a closed mind in the scientific community. We simply decree it is the largest preventable.'" hence, what do we do? We gather together, form a wedge, and move forward to, if you will, stamp out smoking. We move down the line of prohibition.

Again, I am just, as a scientist, inquiring how well conceived that is. Is it the largest preventable? The U.S. Congress is proclaiming it is. If I were in the medical sciences, as I translate my knowledge from what I learned in the behavioral sciences, I would be very reluctant to publicly say, "No question about it. It is 'the largest preventable.'" I query whether the scientific conclusion of that is true, and it seems to me you are bearing me out. You are saying, "Well, it's true, Senator," in somewhat of a speculation of theory, and so on and so forth, "but we couldn't really say categorically that it is so."

But yet these findings are categorical findings. "The Congress finds that." We cannot have it both ways, it strikes me.

Dr. LeMaistre. With all due respect to your argument, Senator, I don't think I said the word speculation. I think this is the judgment by the——

Senator East. You used the word "theoretical," which would imply something less than——

Dr. LeMaistre. Theoretical is far from speculation. I think the point is that, in theory, a lot of things can be postulated; they are not speculated.

Senator, let me see if I can put in perspective the only area in which I think you and I are in agreement. I do think that is a judgment call.

Senator East. Not a scientific finding; a judgment call.

Dr. LeMaistre. I think it is based on scientific findings, Senator. The point I am driving at is, the mass of scientific evidence is weighted in making that call. Similarly, I cannot give you today a complete definition of the way aspirin works, but that is not going to keep me from taking aspirin when I have a headache.

Senator East. But what evidence do we have that it is preventable, I mean in terms of ease of eliminating it more than any of these other areas? That is all I want to know.

Dr. LeMaistre. I think the most overwhelming part of this is that 18 million Americans quit in 1965, as I testified just a few moments ago, with the publication of a single report. There has been a decrease in the taking up of the habit through educational activities. If you asked, would the behavioral scientists come together and say that they can do this or they can do that, either in encouraging people to give up cigarette smoking or prevent them from starting, I am quite sure that there would be a wide range of opinions. But, I think the consensus would be that education—and you said it yourself—comprehensive health education—of which smoking information is only a part, dealing with the consequences of other substances, like salt, clearly know plays a role in health determination in our society.

I would favor very much the kind of educational program that you propose for young people so that they might have all of the facts in all areas and make their own choices about their lifestyles. But the one key, I think, in this, Senator, is that industry has not, despite the fact that there have been pleas for a safe cigarette since 1964, ever successfully marketed a safe cigarette. There have been cigarettes brought out made out of cabbage and other nontobacco substances that were not acceptable to the American public, apparently, in that they did not seem to survive and sell; there have been all sorts of other so-called safety mechanisms introduced. The filter itself, in my opinion, was an attempt to try to make a cigarette safer. But, despite all the scientific evidence which I am aware of, there is no safe cigarette today.

So sitting and hoping for that is not wise. I would condone further research on the safe cigarette and hope that it would by successful. But, in the meanwhile, I think we have to go on the assumption that that is not likely to happen tomorrow, and we do need to put forward information to the American public that they have every right to know.

Senator East. I am just concerned that we not call assumptions, suppositions, and wishes facts, and I fear in this area we are doing that, and I fear in these findings we are doing that, and I fear this, "largest preventable cause," that is, I think, a statement of wish or conclusions, surmise, supposition. There may be some validity in it, but I am just curious about the scientific community too quickly giving the mantle of scientific base to what I think, in the real world of scientific integrity as I understand it, could just simply not be demonstrated, and we might as well admit it and thereby have greater credibility in the long run than when we make these grandiose claims.

I mentioned earlier about nitrites and saccharin and so on. We are throwing out one scientific finding today and, 3 months later, we are pulling it back, or 1 year later or 2 years later, and finally, what the public says is, those fellows don't know what they're talking about, because they really don't have, quote, scientific findings; what they have are general opinions. You used the word "judgments" about things, and they are communicating them out. But all of that is packaged in the terms of these findings. So many different mice, what, some 800 mice had so much saccharin. Oh, it sounds like, oh gosh, these mice drank all these soft drinks and they got cancer, a terrible thing. Well, it turned out it had absolutely no correlation to genuine human health problems, and so it goes—nitrates, and so on and so forth.

Coming out of the behavioral and social sciences, I just want to make sure that—I certainly do not like to see our profession too quickly run into the political fray, and I hope that the scientific community of this country, as zealous as it may feel in terms of its own political judgments or preferences, and they are free to work with those, does not bring the mantle of science in and grace it over this whole problem when, in fact, scientific findings will not bear it out.

Thank you, Mr. Chairman.

Dr. LeMaistre. Mr. Chairman, if I may, I think Senator East has an extremely important point in terms of carcinogenic agents and the consequences of Congress action based on the Delaney clause which overstresses certain potentially misleading criteria for screening those possible agents.

That causes the American public, to worry about cranberry juice and a lot of other things that are not high priority risks in cancer causation. That false worry does diminish appropriate concern over cigarettes. The very credibility of the arguments about cigarettes is damaged by those rather cursory studies often used in claiming for some substances a high risk in cancer. You mentioned nitrates and several other things this morning.

I have the same concern you do in that regard, but I have no concern whatsoever for the overwhelming fact that the evidence is in on cigarettes and that it does lead to proper scientific conclusions and, in my opinion, the judgment calls that we were discussing are based on those proper scientific conclusions.

But I think there is a wide disparity between the consequences of the Delaney clause and the consequences of information gathered on the hazards of tobacco over the past 20-some-odd years.

Thank you, sir.

The Chairman. Well, thank you, Dr. LeMaistre. We are very appreciative that you would come and give us the benefit of your testimony.

Dr. LeMaistre. Thank you, sir.

The Chairman. Thank you.

Our fourth and final panel consists of representatives from the Tobacco Institute. We will hear from Mr. Edward A. Horrigan, the chairman and chief executive officer of the R. J. Reynolds Tobacco Co., and chairman of the executive committee of the Tobacco Institute.

As I understand it, Mr. Horrigan is accompanied by two scientists, Dr. Paul Miniard from Ohio State University and Dr. Hans Eysenck from the University of London.

Thank you for joining us today, Mr. Horrigan. We are glad to have you here.

STATEMENT OF EDWARD A. HORRIGAN, JR., CHAIRMAN AND CHIEF EXECUTIVE OFFICER, R. J. REYNOLDS TOBACCO CO., ON BEHALF OF THE TOBACCO INSTITUTE, ACCOMPANIED BY PAUL MINIARD, ASSISTANT PROFESSOR OF MARKETING, OHIO STATE UNIVERSITY; HANS EYSENCK, PROFESSOR OF PSYCHOLOGY, INSTITUTE OF PSYCHIATRY, UNIVERSITY OF LONDON; SHELDON C. SOMMERS, SCIENTIFIC DIRECTOR, COUNCIL FOR TOBACCO RESEARCH

Mr. Horrigan. Good morning, Mr. Chairman.

As the chairman has mentioned, my name is Edward A. Horrigan, and I am chairman and chief executive officer of R. J. Reynolds Tobacco Co., but I am appearing here this morning in my capacity as chairman of the Executive Committee of the Tobacco Institute.

Joining me this morning for any questions that might be asked following this very brief summary will be Mr. Paul Miniard, assistant professor of marketing at Ohio State University; Dr. Sheldon Sommers, scientific director of the Council for Tobacco Research; and Hans Eysenck, professor of psychology at the Institute of Psychiatry, University of London, and psychologist to the Maudsley and Bethlehem Royal hospitals in London.

Again, given the time constraints, if necessary with respect to your questions, I may also call upon Dr. George Schafer, medical director of the Tobacco Institute; Mr. Horace Kornegay, chairman here of the Tobacco Institute and Sam Witt, who is vice president and general counsel of R. J. Reynolds Tobacco Co. and is seated directly behind me.

We are here today to express the tobacco industry's position on S. 1929, the Comprehensive Smoking Prevention Education Act.

We are firmly opposed to this legislation because we feel that it is bad legislation for the American public and for our industry. We feel that it is unnecessary and ineffective and that the scientific findings cited in the bill in support of its proposals are unsubstantiated and incorrect.

From this committee's perspective, this bill may well be counterproductive because there is significant evidence, from the standpoint of behavioral science, that its actual result may well be to lessen awareness of cigarette health warnings.

A number of very broad, sweeping statements relating to smoking and disease are included in the particulars of this bill. Certainly there are questions raised by statistical associations which must be answered, and our industry is providing significant funding—more annually than all of the private health agencies combined—to support unrestricted research to find those answers.

But the truth is that after three decades of investigation, no causal link between smoking and disease has been established. All of the statements presented as established fact in this bill have been challenged by research findings of many eminent scientists, and I submit that their findings should be considered in your deliberations.

As you know, this bill is very similar to H.R. 5653, the House of Representatives bill which, although broader in its approach, seeks to achieve basically the same goals. Just last Friday, I along with expert witnesses from several disciplines presented the industry's views in opposition to the House bill to a subcommittee headed by Representative Waxman.

At that time, there were over 30 highly qualified and respected scientists ready to appear to challenge the scientific findings of that House bill, which are very similar to those in your bill, S. 1929. I request at this time that their written submissions to the House subcommittee be entered into the record of these proceedings.

I would also add that the time you are devoting to these hearings, to hearing our industry's position, is really far too little in view of the complexities of the issues. Far more time, rather than the very brief period we have here this morning, would be required to cover only the medical and scientific questions in regard to this bill.

In the House subcommittee hearings Friday, we were allowed only a day to cover the issues, which was still far short of the time that was needed. Nevertheless, we will do our best here in the time that has been allotted.

In the South where I now live, there is a saying, "If it ain't broke, don't fix it." I submit that this bill is an unnecessary attempt to change a system that the facts demonstrate is indeed working.

The evidence shows that over 90 percent of the American public is aware of the claim that smoking is harmful, the message that is contained in the present health warning required on every cigarette pack and ad.

This awareness level is virtually, if not totally, unprecedented in comparison to the awareness of the major issues facing this Nation. The facts demonstrate that the Federal Cigarette Labeling Act is working, that the public has been made aware of the claimed health hazards of smoking, and that people are in a position to make a free and informed choice of whether or not to smoke.

Why, then, should it be necessary to substitute a questionable, unproven system for the present law?

There is also strong evidence from the standpoint of behavioral science, based upon the evidence in the studies by the FTC staff in support of rotating warnings, that such a system might in fact erode awareness.

Additionally, I will call your attention to the fact that the Swedish Government, upon whose system this proposal is modeled, indicates and reports that since their system was instituted back in 1977, cigarette consumption has risen annually.

The system of rotating health warnings on cigarette packages proposed by this bill is also technically unworkable and ill-conceived. There are now approximately 200 cigarette brands and brand styles on the market, with new brands being added regularly and old ones being withdrawn. There exist vast differences in sales volume and advertising patterns. Therefore, it would be virtually impossible for cigarette manufacturers or the Federal Trade Commission to insure compliance with this proposed system.

Five rotating warnings required to appear an equal number of times during a 15-month period, with the added provision that at any time each of the five warnings must appear on 15 percent of all cigarette packs and ads, would create chaos.

The bill's requirement of disclosure of tar, nicotine, and carbon monoxide levels on packages and in our advertising is unwarranted. Tar and nicotine levels have been readily available to smokers in every cigarette advertisement since 1970.

With respect to carbon monoxide, the written submissions of scientific experts provided to you will show that carbon monoxide exposure through cigarette smoke has not been shown to be a health issue. Furthermore, there is no generally accepted method of carbon monoxide measurement which would make any data that we would present on our packages and in our ads meaningful.

Next, the provision requiring disclosure of ingredients is unnecessary and ill-conceived. Cigarette manufacturers do use a variety of ingredients to enhance flavor, appearance, and to preserve shelf life. These ingredients are among each manufacturer's most closely held trade secrets. The bill provides that such information should not be disclosed. Unfortunately, despite the good intention of the authors of the bill, those provisions will not prevent unauthorized leaking of these critical trade secrets. On numerous occasions, our industry has been the victim of such unauthorized disclosures of confidential information that we have furnished various Government agencies.

We have been, and we continue to be, engaged in constructive and fruitful discussions with Assistant Secretary Brandt and other HHS officials on this matter, with a view to making available necessary information to that agency under appropriate procedures and safeguards.

Secretary Brandt himself has recently stated that he is pleased with our industry's cooperative spirit and that he is confident that substantial future progress can be made in resolving this question.

This present bill, though, would short circuit this effort to resolve any legitimate concerns. In other words, the effect of this bill would be to "shoot first and ask questions later."

In summary, we are opposed to this bill for many reasons, but key among them are the following: In effect, we believe it will freeze science in its tracks because it may well divert scientific talent and resources from the basic research necessary to resolve the questions of smoking and health, and because it attempts to settle by Congressional edict continuing medical and scientific controversies.

It also seriously erodes the principle of free choice in our democratic society. In denying a person's right to reject official information, this bill betrays its fundamental prohibitionist motives. It says, in effect, that Americans are expressing their basic freedom of choice in rejecting the arguments of antismoking activists and that the Government finds this unacceptable; therefore, steps must be taken to make Americans conform and to encourage the prohibition of smoking. I do not believe that the American people will accept such a rationale.

Mr. Chairman, I will conclude by saying once again that we regret that we have only been given such a short period of time to address these very key issues which do deserve much more lengthy and indepth consideration. I will end my testimony at this time with the request that my formal written statement, with all attachments, be entered into the printed record of these proceedings. I hope that each member of the committee will read my written submission and those of the expert witnesses very carefully in order to gain an accurate picture of the facts in support of our position on this bill.

Thank you, sir.

The **Chairman**. Thank you, Mr. Horrigan. Thank you for your testimony.

I appreciate your concern that these hearings do not provide adequate time to cover all of the issues related to smoking and health, and therefore all of your statements and the statements of any of your associates here today will be incorporated in the record in full, and we will be pleased to accept whatever additional written testimony you would like to have included in the record, so we will keep the record open for a limited period of time so that you can submit any other additional testimony.

[The prepared statements of Mr. Horrigan, Dr. Eysenck, and Dr. Miniard, and the biographical sketch of Dr. Blackwell follow:]

Statement of Edward A. Horrigan, Jr.
on S. 1929 before the Senate Committee
on Labor and Human Resources
(March 16, 1982)

My name is Edward A. Horrigan, Jr. I am Chairman and Chief Executive Officer, R. J. Reynolds Tobacco Company.

I am also the Chairman of the Executive Committee of The Tobacco Institute, an association of tobacco manufacturers with headquarters in Washington, D. C.

We are here to express our industry's position on S. 1929, the Comprehensive Smoking Prevention Eduction Act.

We are firmly opposed to this legislation because we feel it is bad legislation for the American public and our industry. We feel it is unnecessary and ineffective, and that the scientific findings cited in the bill in support of its proposals are unsubstantiated and incorrect. From this committee's perspective, this bill may well be counterproductive because there is significant evidence from the standpoint of behavioral science that its actual result may well be to lessen awareness of cigarette health warnings.

A 1981 Gallup survey, reported by Secretary Schweiker to a large assembly of voluntary health organizations in November of that year, found that "Ninety percent of the population agrees that cigarette smoking is harmful."

In his 1979 report, the U.S. Surgeon General said that "notable changes had taken place in public awareness of claimed smoking hazards and expressed doubt that a higher level of awareness could have any effect on smoking behavior." We share this skepticism.

Indeed, the level of awareness about smoking and health far exceeds public awareness of most, if not all, of the major contemporary issues facing this nation.

Let me provide some examples: A 1980 Gallup poll revealed that less than 25 percent of the public knows what the First Amendment is or what it deals with. Other recent national surveys reveal that nearly 25 percent do not know what happened at Three Mile Island; 36 percent are not aware that the United States must import oil to meet its energy needs; 45 percent do not know that automobiles are the major source of air pollution; and one-third do not know whether the Federal budget is balanced.

By contrast, at least 90 percent of the public is aware of the allegations that smoking is dangerous to health. This level of awareness demonstrates the success of the current Congressionally mandated warning statement, as well as the efforts of public and private organizations.

The Federal Cigarette Labeling and Advertising Act is working. The facts clearly show that the public has been made aware of the so-called health hazards of smoking, and that people are in a position to make a free and informed choice on whether or not to smoke.

Given this fact, why substitute a novel and unproven system for the present law.

I submit that this bill will have little, if any, impact following almost three decades of government and private warnings, the Surgeon General's statements on cigarette packages and advertising, and broad dissemination of anti-tobacco propaganda through the public media.

The fact that millions of people choose to continue to smoke, despite the almost universal awareness of the allegations regarding smoking and health, is frustrating to anti-smoking activists and advocates. And it is our belief that this frustration has led them to conclude that those who reject anti-smoking arguments and continue to exercise their freedom of choice are uninformed. Therefore, they seek measures, such as this bill, designed to force smokers to conform, and to encourage prohibition of smoking.

Let me take a few minutes to review some of the premises used to support this bill and the charges leveled at the tobacco industry.

A number of very broad, sweeping statements relating to smoking and disease are included in the particulars of the bill. Of course, there are questions raised by statistical associations relating to smoking and disease which must be answered. But the truth is that all of the statements that are presented as established fact in this bill have been challenged by the research findings of many eminent scientists, and I submit that their findings should be considered in your deliberations.

As you know, this bill is very similar to H.R. 5653, a House of Representatives bill which, although broader in its approach, seeks to achieve basically the same goals. Just last Friday I, along with expert witnesses from several disciplines, presented the industry's views in opposition to the House bill to a subcommittee headed by Representative Waxman. Also, at that time, there were some 30 highly qualified and respected scientists ready to appear to challenge the scientific "findings" of the House bill which are very similar to those in S. 1929. Only a handful of these scientists were heard; the others were just permitted to file statements. I request at this time that the submissions to the House Subcommittee of all of these scientists be entered into the record of these proceedings.

I would also add that the time you are devoting to hearing our industry's position is far too little in view of the complexities of the issues. Far more time, rather than the brief hour we have been allowed today, would be required to cover only the medical and scientific questions in regard to this bill. In the House subcommittee hearings, we were allowed only a day to cover the issues, far short of the time needed. Today, we will do our best in the time allotted.

In 1969, when the Federal Cigarette Labeling and Advertising Act was amended, the Commerce Committee of the House of Representatives held extensive hearings over a period in excess of two weeks. It heard testimony from Members of Congress, state officials, government health and regulatory agency officials, voluntary health organizations, and numerous expert witnesses in the fields of medicine, biomedical research, statistics, and other scientific disciplines.

On the basis of these extensive hearings, the House Commerce Committee concluded that "nothing new has been determined with respect to the relationship between cigarette smoking and human health since its hearings in 1964 and 1965."

The committee went on to say that "the arguments pro and con with respect to cigarettes are the same now as then, though supported by a larger statistical base." That conclusion is as true today as it was in 1969.

After three decades of investigation and millions of dollars invested by the government, the tobacco industry and other private organizations, the smoking and health controversy remains unresolved. The net result of all of this effort has been that no causal link between smoking and disease has been established. That is not merely the opinion of tobacco industry executives. That is scientific fact readily available to anyone willing to make an objective, unemotional study of the existing evidence.

Yet, consider some of the broad, categorical statments on smoking and disease in this bill. What credibility can such statements have in view of the millions of people who have smoked throughout their lifetimes and not incurred any of the ailments mentioned, and the millions who have never smoked and who have incurred these ailments?

I should add that the tobacco industry is recognized as a leader in seeking the answers to the questions regarding smoking and health. In the past three decades, the industry has committed, without fanfare, over $100 million for unrestricted, independent research into smoking and health issues. Our spending has been greater than the tobacco-related research expenditures of all of the voluntary health agencies combined.

I submit that one of the effects of the bill would be to do significant harm to the scientific effort to resolve these questions. As worded, the bill purports to settle by Congressional edict medical and scientific controversies that are still evolving. Therefore, it may divert scientific talent and resources from the basic research necessary to solve the enigmas of chronic disease.

We are aware that there have been some efforts to build support for this bill with claims that its provisions would serve as a deterrent to smoking among young people and that industry advertising and promotional practices are intended to encourage youthful smoking.

Such charges are without foundation.

On March 5, two witnesses speaking in support of the House bill -- Mr. Keeshan and Mr. Forsyth -- both acknowledged that peer pressure, and not our advertising, provides the impetus for smoking among young people.

The written submissions of expert witnesses we are providing to this committee clearly point out that the provisions of this legislation can in no way be justified by the emotionally appealing but unsupported assertion that cigarette advertising encourages a youngster to smoke.

Smoking is an adult practice to be considered only by those mature enough to make an informed decision.

In 1963, for example, cigarette companies stopped all advertising and promotional activities in school and college publications and on campus. We also stopped using celebrities and sports figures in advertising.

In 1964, we adopted a cigarette advertising code prohibiting advertising, marketing and sampling directed at young people. Even though the administrative provisions are no longer in effect, each company still adheres to the principles of this code. A summary of these principles and a copy of our Cigarette Sampling Code is being submitted with this document for the record.

In 1969, we offered to cancel all radio and television advertising because of broadcast's unique reach to young people, and in 1971, pursuant to federal legislation, left the broadcast media.

Our advertising is targeted at smokers and is intended to encourage switching from competitive brands. The available evidence clearly shows that our advertising is not designed to attract new smokers of any age and is not having that effect. The same Gallup poll I mentioned earlier also reported that the percentage of smokers found was the lowest ever recorded by that organization.

Looking at the broader picture regarding cigarette advertising, the record clearly shows that our industry has been responsible in its practices.

o In 1954, to meet public demand, we began to advertise low "tar" and nicotine cigarettes.

o In 1960, after the FTC stated that it had determined that such advertising could be construed as a health claim, we voluntarily agreed to eliminate from cigarette advertising all references to "tar" and nicotine.

o In 1966, when the FTC reversed its position, we agreed that cigarette advertising would disclose "tar" and nicotine content.

o In 1967, we began a continuing program of scientific and technical cooperation with the FTC with respect to "tar" and nicotine testing.

o In 1970, we began to include in our brand advertising the FTC "tar" and nicotine measurements.

o In 1971, we volunteered to depict the cigarette package in all advertising in such a way as to display legibly the warning label.

o In 1972, we entered into an agreement with the FTC on uniform terms of conspicuously displaying the warning label in advertising.

o In 1981, we reached an agreement with the FTC on an increase in the size of warnings.

In short, our industry has acted responsibly in the past and we see no reason anyone should feel that we will not continue to do so in the future. Nor do we see why this bill is needed to regulate further our advertising practices in any way.

Getting back to the specific provisions of the bill, the labeling requirements proposed are unworkable.

The bill directs the FTC to establish a rotational warning procedure which can at best be described as a "Rube Goldberg" contraption. Every brand of cigarettes would have to carry each of the five required warnings an equal number of times during a 15-month period. Then there is the added provision that at any time each of the five warnings must appear on "at least 15 per centum of all cigarette packages and advertising."

In December 1981, the FTC reported "tar" and nicotine data on 200 cigarette brand styles. New brands are frequently introduced and old ones withdrawn. It is highly unlikely that the FTC or any other agency will be able to develop anything but a purely arbitrary formula for rotating the warning statements among the various brands.

Even if a rational formula could be devised, how could we make sure that each warning statement is presented to the public an equal number of times? Sales volume and advertising for each brand vary greatly. Equalizing warning statements among all brands will have no relationship at all to the number of times that each statement is exposed to the public.

Technical complexities aside, there is no reason whatsoever to believe that the proposed rotational warnings would be any more effective in increasing public awareness than the present statement. The proposal is based on a recommendation of the FTC staff, which in turn was based on some undisclosed preliminary research regarding a rotational warning system used in Sweden.

The FTC staff admits that the "effectiveness" of Sweden's system cannot be measured, and Mr. Waterson, an advertising expert, on March 5, presented persuasive testimony to the House subcommittee to the effect that this system is not working to reduce consumption in Sweden. While the FTC staff concedes that the "effectiveness" of the Swedish system cannot be evaluated, the fact is, that according to the Swedish government, cigarette consumption has risen each year since the new system was implemented in 1977. I also call your attention to the fact that the rotational warning system in Sweden was part of a total program designed to abolish smoking, which is the ultimate goal -- despite denials -- of many of the anti-smoking organizations which support this bill.

There is also strong evidence from the standpoint of behavioral science, based upon the evidence available in the studies cited by the FTC in support of rotating warnings, that such a system might, in fact, erode awareness.

In light of these facts, the obvious question arises: Is the proposed rotational system necessary or appropriate? We are convinced that the answer is that it is neither.

Also unwarranted, in our opinion, is the bill's requirement for disclosure of "tar," nicotine, and carbon monoxide levels on packages and in all advertising.

As indicated earlier, since 1970 cigarette manufacturers have voluntarily disclosed the "tar" and nicotine levels in cigarette advertising. This information is widely available to the public. Smokers who choose their brand on the basis of "tar" and nicotine levels can readily obtain this information from cigarette advertising.

As a matter of fact, the average "tar" yield of cigarettes sold in this country has dropped from 38 milligrams in 1956 to 12.6 milligrams in 1981. Almost 70 percent of all cigarettes sold are in the low "tar" category.

With respect to carbon monoxide, there is no purpose to be gained by the determination or publication of yields. As the scientific record will show, the conclusion that exposure to carbon monoxide from cigarette smoking is significant in terms of health is unwarranted. Furthermore, no single method of carbon monoxide measurement in cigarette smoke has gained general acceptance in the scientific community. Therefore, any data presented, by any method, will be subject to question. These issues should be resolved in the scientific community. We firmly believe that a required disclosure of carbon monoxide yields is ill-conceived.

The provision requiring disclosure of ingredients is also unjustified. Cigarette manufacturers use a variety of substances in their products to enhance flavor and appearance and to preserve shelf life. The identity of ingredients used by each manufacturer is among the most closely guarded of their trade secrets, for such ingredients play a substantial role in maintaining consumer acceptance.

The bill provides that such information should not be disclosed. Unfortunately these provisions will not serve to prevent the unauthorized leaking of these most important trade secrets. On numerous occasions our industry has been the victim of unauthorized disclosures of confidential information furnished government agencies.

The basic defect in this provision is that it is an attempt to provide a quick and easy "solution" to a problem that has not yet been adequately defined.

We have been, and continue to be, engaged in constructive and frutiful discussions with Assistant Secretary Brandt and other HHS officials on this matter with a view to making available necessary information to HHS under appropriate procedures and safeguards. Secretary Brandt has recently stated that he is "pleased with" our "cooperative spirit" and that he is "confident that substantial future progress can be made" in resolving this question. The present bill would short-circuit this effort to resolve any legitimate concerns. In other words, the effect of the bill is to "shoot first and ask questions later."

In conclusion, we are firmly opposed to this legislation because we believe it to be unnecessary and, most importantly, because the medical and scientific assumptions or "findings" underlying it are incorrect and unsubstantiated.

We are not opposing the bill because we wish to exploit the youth market as some have falsely charged; nor are we opposing the bill because of the potential costs of complying with its requirements.

We oppose this bill because, despite the appearance of good intentions, this is bad legislation -- not only for our industry but the American public as well.

This bill freezes science in its tracks. It purports to settle by Congressional edict medical and scientific controversies that are still evolving, and thereby it may divert scientific talent and resources from the basic research necessary to solve the enigmas of chronic disease.

This bill also seriously erodes the principle of free choice in a democratic society. It says, in effect, that if you don't conform, you are uninformed, and that the government must take corrective action. In denying a person's right to reject "official" information, the bill betrays its fundamental prohibitionist motives. The proponents of the bill object to the fact that Americans are expressing their basic freedom of choice in rejecting the arguments of anti-smoking activists; the sponsors find this independence unacceptable, and therefore, propose steps intended ultimately to result in the prohibition of smoking.

We are a responsible and concerned industry, manufacturing a lawful product which provides pleasure and satisfaction to millions. Our industry contributes more than $57 billion annually to the Gross National Product and generates $22 billion in federal, state and local taxes of all kinds.

This proposed legislation is an unwarranted intervention by the federal government into the private lives of its citizens, and a thinly veiled effort further to harass and ultimately eliminate an important American industry.

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Mr. Edward A. Horrigan, Jr., Chairman of the Executive Committee of the Tobacco Institute, is the Chairman and Chief Executive Officer of R. J. Reynolds Tobacco Company. He is also an Executive Vice President and Director of his parent company, R. J. Reynolds Industries.

He is a graduate of the University of Connecticut and the Harvard Business School's Advanced Management Program.

During the Korean conflict, he served as a regular Army infantry officer and received the Silver Star, Purple Heart and Combat Infantry Badge.

He entered the business world in 1954 and was first employed by Procter & Gamble in sales management and later rose to Division Vice President at Thomas J. Lipton, Inc. before joining the Buckingham Corporation in 1973 as Chairman of the Board and President. The Buckingham Corporation is one of America's leading importers of wine and spirits. He joined R. J. Reynolds Tobacco International in 1978 as Chairman and Chief Executive Officer and was promoted in January 1980 to Chairman, President and Chief Executive Officer of the U. S. operations, R. J. Reynolds Tobacco Company. Later that year he was named to the R. J. Reynolds Industries Board of Directors and in 1981 was elected to Executive Vice President of the parent company with responsibility for Reynolds' worldwide tobacco operation.

During the course of his business career, Mr. Horrigan has received many awards for his business and philanthropic accomplishments. In 1981, he received the prestigious Horatio Alger Award and the American Jewish Committee's award. He is also a recipient of the Pop Warner All American Award for service to youth and received the Distinguished Alumni Award from the University of Connecticut.

Mr. Horrigan serves as a Director at Salem Academy and College and on the Board of Visitors of the School of Business Administration at the University of Connecticut. He is also currently serving as Chairman of Winston-Salem State's first capital campaign drive and is a Honorary Board Chairman of the Touchdown Club of America.

Principles Governing
Cigarette Advertising and Sampling

These advertising principles apply to all forms of advertising including vehicle decals, posters, pamphlets, matchbook covers, and point of purchase materials in the United States, Puerto Rico, and U.S. territorial possessions.

1. No advertising shall appear in publications directed primarily to those under 21 years of age, including school, college or university media (such as athletic, theatrical or other programs), comic books or comic supplements.
2. No one depicted in cigarette advertising shall be or appear to be under 25 years of age.
3. Cigarette advertising shall not suggest that smoking is essential to social prominence, distinction, success or sexual attraction, nor shall it picture a person smoking in an exaggerated manner.
4. Cigarette advertising may picture attractive, healthy looking persons provided there is no suggestion their attractiveness and good health is due to cigarette smoking.
5. Cigarette advertising shall not depict as a smoker anyone who is or has been well known as an athlete, nor shall it show any smoker participating in, or obviously just having participated in, a physical activity requiring stamina or athletic conditioning beyond that of normal recreation.
6. No sports or celebrity testimonials shall be used or those of others who would have special appeal to persons under 21 years of age.
7. Persons who engage in sampling shall refuse to give a sample to any person whom they know to be under 21 years of age or who, without reasonable identification to the contrary, appears to be less than 21 years of age.
8. Sampling shall not be conducted in any public place within two blocks of any centers of youth activities, such as playgrounds, schools, college campuses, or fraternity or sorority houses.
9. Persons who engage in sampling shall not urge any adult 21 years of age or over to accept a sample if the adult declines or refuses to accept such sample.

LUNG CANCER, CORONARY HEART DISEASE AND SMOKING

By H. J. Eysenck, Ph.D., D.Sc.
Professor of Psychology
Institute of Psychiatry
University of London

March 10, 1982

Statement of Professor Hans J. Eysenck

I am Hans J. Eysenck, professor of psychology at the Institute of Psychiatry, University of London and psychologist to the Maudsley and Bethlem Royal hospitals in London.

I received my Ph.D. in 1940 and my D.Sc. in 1964, both from the University of London. I was Senior Research Psychologist at Mill Hill Emergency Hospital from 1942 through 1946. In 1949 and 1950 I was a visiting professor at the University of Pennsylvania in Philadelphia. Between 1950 and 1954, I was a Reader in Psychology at the University of London's Institute of Psychiatry. In 1954 I was a visiting professor at the University of California at Berkeley.

I am a Fellow of both the British Psychological Society and of the American Psychological Association.

I have founded and edited three psychological journals, and I am on the editorial boards of some 15 other international psychological journals. I have written or edited for publication approximately 35 technical books and over 600 articles dealing with various aspects of the psychological field, particularly with respect to personality, intelligence, behaviour therapy and behavioural genetics. I have conducted research in the area of smoking for over 20 years and have authored two books, the most recent of which is entitled The Causes and Effects of Smoking, as well as numerous articles on this subject.

A widely accepted theory asserts that cigarette smoking causes lung cancer, coronary heart disease, and many other diseases with which it is statistically linked. It is not always realized that (a) such a theory is far from proven, and is beset by many anomalies and doubts, and that (b) there is an alternative theory which is based on undeniable facts which are not explained by the causal theory. The present position seems to be that either theory may explain the tragic incidence of lung cancer and coronary heart disease (to which this brief account will be restricted), or that both may be needed to complement each other.

There is agreement that smoking is neither a necessary nor a sufficient cause of lung cancer. Of 100 heavy smokers, less than 10 will develop lung cancer; hence smoking is not a sufficient cause. And of 100 people who develop lung cancer, approximately 10 will be non-smokers: hence smoking is not a necessary cause. This simple fact (the precise numbers differ of course from country to country, but indicate the correct order of magnitude) suggests that the scientific proof for any particular theory will be difficult to arrive at, and that any such theory will almost certainly be complex and multi-faceted.

Much of the evidence cited in favour of the causal theory is statistical, but many statisticians have severely criticized the evidence on statistical grounds. Such suggested proofs as the correlation between smoking and lung cancer within a given country, or between lung cancer and number of cigarettes smoked between countries, are evidence of correlation, not of causation; one of the first lessons the budding statistician learns is that correlation does not imply causation. (There is a very high correlation between countries linking meat eating and cancer of the large intestine, yet we do not conclude that eating meat causes cancer of the large intestine!). Hence this method of demonstration, while suggestive, is far from compelling. This would be so even if the figures usually quoted could be taken seriously; however, there are good reasons for doubting their accuracy.

The figures quoted are based on clinical diagnosis of lung cancer, but these are very unreliable and imprecise. If we take as our criterion autopsy data, and compare these with routine diagnosis, we find that prior to World War 1, out of 100 people found on autopsy to have died of lung cancer, only 3 were so diagnosed. This is typical of the very obvious under-diagnosis of lung cancer then prevalent. In recent years, exactly the opposite has been found, namely an over-diagnosis of lung cancer of up to 200% and more! Whether these changes in diagnostic preference are completely responsible for the alleged tremendous increase in lung cancer over the years or not, and whether it may in part account for the observed correlation between lung cancer and smoking, it is impossible to say; all we can say is that with the basic data so completely unreliable, the statistics based on them are suspect.

Another important point concerns the isolation of smoking from other, correlated habits, such as drinking, living it up, staying out late, wenching, etc., i.e. a certain style of life the totality of which may increase the "rate of living", so that smokers are biologically older than non-smokers at a given age, for reasons only partly involved with smoking. Non-smokers are different types of persons from smokers, are generally more self-protective, and the personality traits and habits thus linked with non-smoking may be more relevant to the longevity of non-smokers than their refusal to smoke.

It is often suggested that sex differences, with males showing more lung cancer, are the product of the tendency of males in the past 50 years or so to smoke more. However, as several authorities whom I quote in my book have pointed out, similar sex ratios to those observed now were found before cigarette smoking became popular. Again, it is found that changes in the rate of increase of lung cancer diagnosis occurred *simultaneously* for men and women, although the women, who took up smoking much later than men, should have shown these changes at a much later date than men.

If the causal theory is true, then we would expect a definite dose-response relationship; in other words, the heavy smoker should be stricken with cancer *earlier* than the light smoker. Yet the amount smoked makes no appreciable difference to the mean age at which the person is reported first to the clinic. Again, inhalation should make lung cancer much more likely than smoking without inhaling, yet the figures show if anything an opposite trend. These two observations are difficult to reconcile with the causal theory of smoking.

The most impressive evidence for the causal theory has been the report that physicians who gave up smoking showed less lung cancer than members of the general public who continued to smoke. Thus, it might appear that giving up smoking has saved the lives of those who did so. But this proof is only acceptable if those who continue to smoke and those who later on give up smoking, are essentially identical with respect to their health before some of them gave up smoking. Clearly, if those who later on give up smoking are already much healthier than those who later on continue to smoke, then the final differences in health may be due to the already existing differences before anyone gave up smoking, rather than to the cessation of this habit! But there is good evidence to show that smokers and ex-smokers already differed with respect to their health record before the ex-smokers gave up smoking. Similarly, there is evidence that from the point of view of personality ex-smokers are different from continuing smokers. Thus this alleged proof is based on an erroneous assumption.

These objections to the causal theory, and others made in my book, do not prove the theory to be wrong; they simply argue that it is still only a theory, not a scientific law. More convincing proof is required before the theory can be accorded a more advanced status. But further than that, there are numerous facts suggesting an alternative theory, and these facts cannot easily be integrated with the causal theory. Yet a proper theory demands that attention be paid to all relevant facts, and thus again the causal theory is found wanting.

The alternative theory, first suggested by the eminent geneticist and statistician Sir Ronald Fisher, suggests that genetic factors are important in causing lung cancer; that genetic factors are active in causing people to maintain the smoking habit; and that possibly the same genetic factors may be involved in both these trends, thus producing the observed correlation between smoking and cancer (insofar as such a correlation is real). There is evidence that genetic factors do play a part in the causation of lung cancer; this is not in doubt. I have brought forward evidence (in addition to already very convincing evidence produced by many other people) to show that genetic factors are relevant to the maintenance of the smoking habit. Thus there is evidence for both the assumptions on which Fisher's argument was based.

The origin of the smoking habit, on the other hand, is hardly at all influenced by genetic factors. It appears from our genetic analysis and from the direct study of the problem by Professor Spielberger that the origin of the smoking habit is due to peer pressure; parental influences play a much smaller part, and advertising almost none.

My own contribution has been to suggest that the mediating factor between cancer and smoking may be the personality of the people involved. Thus it is assumed that people of a certain personality are more likely than others to die of lung cancer irrespective of smoking. It is also assumed that people of a certain personality are more likely to smoke than others. There is evidence for both these propositions. My original work with Dr. Kissen, an eminent British oncologist, showed very marked personality differences between lung cancer patients and patients suffering from non-malignant tumours, with the personality assessment made before diagnosis. Since then, a large-scale study in East Germany has replicated our findings (themselves replicated in another study by Kissen), and has found similar personality traits to those characteristic of lung cancer patients in women with cancer of the breast. Other studies, also indicating a relation between lung cancer and personality, are cited in my book.

In a similar way, my early work with Tarrant and Woolf established a correlation between personality and smoking, and many studies in different countries have since confirmed our findings, and added new ones. We may thus say that the fundamental assumption of Fisher's genetic theory have found empirical support, and we may add that there is also some modest support for my own attempt to integrate these two major fields. Unfortunately there has been too little work along these unusual and somewhat unorthodox lines to say that the results are anything more than suggestive, and the theory linking them is still in a very elementary stage; nevertheless, as far as the findings go they support the genetic rather than the causal theory, although they do not necessarily contradict the latter. It seems unfortunate that the premature crystallization of spurious orthodoxies has prevented the genetic theory from attracting sufficient research grants to work it out in sufficient detail, and to carry out the research necessary to put it on a more acceptable footing.

Recently some progress has been made on the theoretical development of the genetic hypothesis by linking it with research on stress, in particular the differential effects of chronic and acute stress, and the "inoculation" theory of stress. However, in the absence of large-scale research into the refinements of this theory, and more widespread familiarity with and criticisms of its details, not too much should be claimed for it other than it presents a viable alternative to the causal theory.

In relation to the causal theories of coronary heart disease (CHD), similar criticisms apply as do in the case of lung cancer. There are considerable unreliabilities in diagnosis; there are large numbers of factors other than smoking which have been associated and which are not usually controlled for in studies of the effects of smoking; inhalers do not on the whole differ from non-inhalers in disease proneness; the statistical relation between cigarette smoking and CHD disappears in many countries, e.g. Finland, Holland, Yugoslavia, Italy, Greece and Japan; there is an absence of dose-response relationship, i.e. there is little or no relation between duration of heavy cigarette smoking and risk of myocardial infarction; and the correlation between number of cigarettes smoked and CHD is not linear; ex-smokers in some studies appear to be safer than non-smokers; some types of CHD, such as angina pectoris (which comprises some 20% of CHD in men) fail to show even a statistical correlation with cigarette smoking; some types of smoking (cigar, pipe) fail to show even a statistical correlation with CHD; etc. These are anomalies or failures of the causal theory which demand an explanation before the causal theory can be accepted. Some of these facts are much more readily explained in terms of a genetic-personality theory; thus the differential effects of cigarette vs. pipe/cigar smoking may find an explanation in terms of the known differences in personality type associated with these different smoking patterns.

The general conclusion would seem to be that in the case of CHD, as in the case of lung cancer, proof for the causal influence of smoking is still lacking and is by no means as clear-cut and decisive as is often alleged. There is evidence in the case of CHD of genetic factors, and there are published correlations with personality; here too there appears an important element of stress determining the appearance of CHD, and stress is intimately linked with personality. No formal theory of genetic determination of CHD has yet been put forward, but it seems likely that such a theory is needed as an alternative (or perhaps as complementary) to the causal theory for an explanation of the many gaps and anomalies in the latter.

One important function of the genetic theory has been that of explaining the reasons why people smoke, and to link these reasons with their differential personality patterns. Work along these lines has had the important effect of suggesting new and improved ways of teaching people to give up smoking. The causal theory of smoking causing disease has nothing to say on this topic. Another important function of the genetic theory has been to suggest better designs for research in this complex field; a good example is the use of the discordant twin method by Cederlof, Lundman and others, i.e. the investigation of the illness patterns of identical twins of whom one smokes, the other not. If this type of research had been carried out on the large and international scale required, instead of investing in the redundant and scientifically not very valuable replication of correlational studies, we would know far more about the relation between smoking and disease than we do now. Such studies allow us to look at environmental factors, including those of smoking, while controlling for genetic factors; this is essential if any convincing results are to be achieved.

In summary I would like to state that the causal theory of smoking as being responsible for lung cancer and coronary heart disease, while it has found strong support, is far from being established, and has many gaps, anomalies and contrary findings to contend with; these are too frequently glossed over and dismissed as unimportant, when in reality they may be found to discredit the causal theory in whole or in part. An alternative theory, based on genetics and implicating personality factors, is much less well developed, more complex, and at present not too well known to oncologists; nevertheless there are many well-established facts which suggest that in part if not in whole it can account for the major findings. At the very least, this alternative theory suggests novel research methodologies which would serve to overcome the difficulties of the older methods and remedy their lack of proper controls. The possibility has also been raised that the two theories may be complementary, rather than opposed to each other; this possibility too should be looked into from the experimental point of view. What is certain is that at the moment no final decision can be made about whether or the degree to which cigarette smoking may cause lung cancer or coronary heart disease, how it interacts with other factors (stress; personality), or how we can best protect the health of our citizens in relation to these diseases. "In ignorance, abstain!" warned the famous French scientist, Claude Bernard; hasty action on the basis of partial knowledge is unlikely to be in the best interests of those most concerned, namely the prospective victims of lung cancer and coronary heart disease.

HANS J. EYSENCK

STATEMENT OF DR. PAUL MINIARD

I am Paul Miniard. I have a doctorate in marketing from the University of Florida, and since last September have been Assistant Professor of Marketing at the Ohio State University. My specialty is the study of consumer behavior, and I have published articles in that and related fields in such journals as the *Journal of Marketing Research*, *Journal of Consumer Research*, and the *Journal of Experimental Psychology*.

For over two years, I have worked with Dr. Roger Blackwell, Professor of Marketing at the Ohio State University and author of, among other things, *Consumer Behavior*, the most widely adopted textbook in the field. I assisted Dr. Blackwell in reviewing and analyzing the cigarette labeling and advertising provisions of S. 1929, the comparable provisions of H.R. 5653, and the FTC Staff Report on the Cigarette Advertising Investigation. On Friday, March 12, Dr. Blackwell appeared before the House Subcommittee on Health and the Environment to provide his analysis of H.R. 5653. He also has prepared testimony concerning S. 1929, but because of a longstanding professional commitment is unable to be here today, and has asked me to present the attached statement.

STATEMENT OF DR. ROGER D. BLACKWELL

My name is Roger D. Blackwell. I am Professor of Marketing at the Ohio State University, specializing in the analysis of buyer behavior and development of marketing strategy. My Ph.D. degree was earned at Northwestern University, with a concentration in consumer behavior. I have authored or co-authored fourteen books and over fifty articles published in professional or business journals that report research that I and others have conducted concerning the communications process, consumer decision processes involved in buying and using goods and services, and variables involved in marketing strategy. One of my most recent books is the fourth edition of Consumer Behavior, published this year. The book describes psychological principles involved in buying and consumption and is the most widely adopted textbook in the field. A complete list of my publications is submitted with this statement.

This is my personal statement and should not be construed to reflect the views of the Ohio State University or any other institution with which I am or have been affiliated.

I have been asked to analyze Section 5 of S. 1929, which would change the present labeling requirements for cigarette packages and advertisements. The rotational system of warning statements proposed by Section 5 of the bill is similar to a recommendation made last year by the Staff of the Federal Trade Commission in a Report on cigarette advertising, and I have evaluated the findings and conclusions of that Report as well.

In my opinion, the change in the warning statement proposed by Section 5 is fundamentally flawed. First, the labeling provisions of Section 5 would replace a highly successful program of informing consumers about the claimed health risks of smoking with a program of unknown and potentially counterproductive consequences. All of the studies conducted about consumer awareness of smoking and health issues lead to the conclusion that people are universally aware of the claims that smoking is hazardous to health. Why abandon this program in favor of a course that is not only uncharted but, as I hope to demonstrate, likely to lead to results quite the opposite of those apparently intended by the bill's sponsors.

The other basic flaw in Section 5 is that the system of rotated warnings attributing specific health problems to smoking will probably lead to one of two unhappy effects: either consumers will erroneously believe that they will personally suffer the specific health problems identified in the warning statements, even though such problems affect only a minority of the smoking and nonsmoking population, in which case the warnings would be deceptive; or consumers will correctly perceive that only a small proportion of the population is at risk from the stated health problems, and will thus conclude that the Surgeon General and other health authorities have now decided that smoking is not hazardous to all people. Since it appears that the specific diseases identified in the proposed warning systems apply primarily to older people and pregnant

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woman, the probable effect of the law would be to reduce drastically the impact of the warning statement on young people.

The rotational warning system proposed in the present bill and the FTC Staff Report apparently is based on the assumption that present public awareness of the claimed health consequences of smoking is "insufficient." Finding 6 in Section 2 of S. 1929 states that "present Federal, State, and private initiatives have been insufficient" in conveying information about the claimed health consequences of smoking to the American public. Similarly, the FTC Staff Report asserts that "additional action designed to provide consumers with more information about the health consequences of smoking is necessary." (Report at p. 21) For several reasons, this assumption is dubious.

In the first place, determination of what constitutes a "sufficient" level of awareness in such a complex area is both difficult and subjective, although it appears to me that, by any standard, the level of awareness about the claimed health hazards of smoking is astonishingly high. A basic question is the amount of information a consumer can reasonably be expected to be aware of in connection with a decision to use any particular product. Many of the questions posed in the surveys cited by the FTC Staff required a detailed scientific knowledge about questions of smoking and health, including a complete awareness of every health problem that has been attributed to smoking, the specific size of the increase claimed in the risk of incurring each problem if one smokes, the percentage of each particular health condition that is attributed to smoking, and the proportion or number of people who die from a given health condition. From the consumer viewpoint, what value is there in possessing such a complex array of information? When one considers the tremendous amount of information to which the consumer is exposed every day, and the fact that consumers do not possess unlimited processing capacities, it clearly would seem more functional for the consumer to retain in memory the overall implication of these numerous bits of information about the claimed consequences of smoking, i.e., that smoking is dangerous. That is precisely the information conveyed by the present Surgeon General's warning statement.

Proper evaluation of the adequacy of consumer awareness also is hampered by the fact that there is no baseline for comparison. For example, how does consumer awareness about smoking and health compare to the information consumers possess about the health hazards attributed to other products such as automobiles, liquor, and hang-gliders? Without such comparison, judgments about the sufficiency of the level of consumer awareness are highly subjective and cannot serve validly as a basis for the far-reaching changes embodied in Section 4 of the bill.

Moreover, an examination of the studies on which the FTC relies for the proposition that consumers are not sufficiently aware of the dangers associated with smoking reveals that those studies are defective in several important respects.

Perhaps most significant is that many of the survey measures assessed beliefs rather than awareness. The distinction between belief and awareness is a critical one given the existing controversy over the health threats presumed to be posed by smoking. Consider the likely situation of a survey participant who recognizes that smoking has been found to be associated with particular health problems but finds the evidence insufficient for demonstrating that smoking causes these health problems. Thus, the person is aware of the claimed link between smoking and some health problems but does not believe that smoking causes the problems. As stated in the 1980 Burke Marketing Research Focus Group Study commissioned by the FTC:

> "Further doubt about the direct relationship of smoking and cancer seems to be related to the fact that these persons had known smokers who had lived long lives without contracting cancer and non-smokers who had suffered from that disease." (Burke Study Analysis at p. 4.)

Many of the measures employed in the studies asked the respondents to indicate their agreement with or the correctness of statements such as "smoking causes X." Respondents who disagreed with these "supposedly true" statements are categorized as unaware. Alternatively, these respondents may be aware of the medical evidence but have concluded that while smoking is "related" to X, it does not "cause" X. Evidence supporting this alternative explanation is provided by the 1980 Chilton study conducted for the FTC Staff. At one point survey participants were asked whether heart disease "had been found to be associated with cigarette smoking." (Question 42e) Only 9.8% of the teenagers and 9.3% of the adults interviewed answered incorrectly (i.e., "No" and "Don't know" responses). Later in the interview, these same persons were asked whether the statement "cigarette smoking is a major cause of heart disease" (Question 52) was true or false. 26.8% of the teenagers and 39.6% of the adults were presumed to be "unaware" of the claim embodied in this statement. Such response variations between questions involving the same disease, but which differ in positing smoking as either the cause of or simply associated with that disease, strongly suggest that many persons classified as "unaware" in fact are aware of smoking's asserted relationship to various health risks. These persons simply do not believe that smoking causes these health problems.

Question wording has long been recognized as a critical area in survey research. The FTC Staff Report acknowledges that ". . . conservative sounding statements have been found to be more likely to generate agreement . . . ." (Report at p. 3-3) By the same token, statements employing extreme wording or phrases are likely to inhibit agreement. Thus, the amount of agreement with the statement "smoking is by far the greatest cause of lung cancer" used in the 1980 Roper Study was probably lower than had the statement been phrased "smoking is the greatest cause of lung cancer." Wording ambiguity can also influence the response patterns to a question. Phrases repeatedly appearing in the Roper Study such as "by far," "greatly increases," and "significantly increases," are very subjective. For example, some people may perceive a 30% risk increase as a significant increase, while others may not.

It is interesting to note that the FTC Report cites evidence that people tend to ignore or discount statistical information in making judgments. (Report at pp. 4-14 and 4-15) Given this evidence, it seems inconsistent to employ measures of "statistical knowledge" for assessing the level of awareness concerning the claimed effects of smoking. Measures of this type, however, were frequently employed as indicators of consumers' awareness about the asserted dangers of smoking (e.g., "What percent of lung cancer cases are caused by cigarette smoking?" - Chilton 1980; "Smokers are at least ten times as likely to develop lung cancer than non-smokers" - Roper 1980).

For these reasons, it would appear that current studies have underestimated consumers' awareness about the health hazards associated with smoking. There is, unfortunately, no way of predicting how much underestimation error exists in these data. But in view of the fact that these same studies consistently report awareness levels in the 80 to 90 percent range, it is fair to conclude that public awareness of the various claims about smoking and health is as a practical matter universal.

It would be wrong to conclude that consumers are not adequately informed about the claimed health consequences of smoking simply because many consumers continue to smoke. The FTC Staff Report states several times that smokers are not as well informed as nonsmokers, implying that such informational differences are responsible for the decision whether or not to smoke. That conclusion is flatly refuted by the 1980 Chilton Study, the very study cited by the FTC Staff to demonstrate the supposed difference in the levels of awareness between smokers and nonsmokers. The conclusion reached by the Chilton Study was as follows:

> "Factual knowledge about the health consequences of smoking was not found to be significantly related to current smoking behavior. No more differences between knowledge levels of smokers compared with non-smokers were found to be significant at the 0.05 level than were to be expected by chance." (page 22)

This important finding that consumer knowledge has no relationship to smoking behavior refutes the notion that people who smoke do so because they are "uninformed" about the claimed dangers of smoking. The lack of a relationship between awareness and smoking also demonstrates that increasing consumers' awareness about the health hazards attributed to smoking is unlikely to influence their smoking behavior. Consequently, to the extent that the present bill is based on a desire to reduce smoking -- and putting aside the question whether behavior modification is an appropriate goal for government in this country -- the warning statements proposed by Section 4 are simply irrelevant.

Given these facts, a change of the sort contemplated by Section 5 should not be undertaken unless there is significant evidence that the proposed system of rotated warnings would better achieve the goal of informing the public. The little evidence that exists not only fails to support that proposition, but in fact contradicts it.

An initial study for the FTC Staff was undertaken in 1980 by Walker Research to assist in the selection of specific warning statements, while another study conducted by Burke Marketing Research examined consumers' recall of various warnings. Importantly, neither study examined the impact of such warnings on consumer awareness. There are accordingly no data to indicate that a rotational system such as that proposed by the bill would meet the objective of providing consumers with "sufficient" awareness about the claimed consequences of smoking, particularly if sufficiency is gauged by the very detailed measures used in the surveys cited by the FTC Staff.

What the studies do demonstrate is that consumers are likely to discount warnings that link smoking to specific health problems. In its Summary of Key Findings, for example, the Burke Focus Group Study states:

> "The messages related to birth control pills and heart attacks tended to confuse the participants, who did not thoroughly understand the synergistic effects which form the basis of the message. These two statements relating to oral contraceptives also had the least personal relevance and were rather easily dismissed as being intended 'for someone else'." (Emphasis added.)

The Study thus concludes:

> "It seems that the birth control message could have relevance to a highly select group of people who could be best reached through very specific media. The message might be lost to the population as a whole." (Burke Focus Group Study Analysis at p. 6.)

That same conclusion appears to apply with equal validity to each of the specific disease warnings contemplated by S. 1929.

Thus, even the preliminary research that has been done tends to support the conclusion that the proposed rotational warnings would be considered irrelevant by some consumers, as compared to the present warning statement that announces to every consumer the Surgeon General's conclusion that "smoking is dangerous to your health."

These findings are particularly significant in view of one of the major premises of the FTC Staff Report, and presumably of the present bill: that consumers should perceive information concerning smoking and health to be personally relevant. Since it is basic to human nature to conclude that risks apply to "the other person," specific warnings that might be more personally relevant to some consumers would by definition be personally irrelevant to most other consumers. For the individual who sees these diseases as unlikely to occur personally, then the proposed new warnings would be less relevant. Such individuals are particularly likely to be young consumers who may be making the decision of whether to smoke or not. While the decision to smoke is not related to advertising, at least in the present situation parents or peers can say to people who are deciding to smoke that "the Surgeon General has determined that smoking is dangerous to your health." Under the proposed new warnings, the logical conclusion would be that the Surgeon General has no longer determined that smoking is generally unhealthy, but only unhealthy for certain older segments of the population or for pregnant women.

As an analyst of consumer behavior and decision making, I am of the opinion that A. 1929 may well have the opposite effect of that which appears to be intended by its sponsors. I am concerned that S. 1929, no matter how laudable the intentions of the sponsors, is not desirable legislation if the goal is to have the general population, and especially smokers, aware of the health hazards that are alleged to occur from smoking.

Dr. Roger Blackwell

Dr. Roger Blackwell is Professor of Marketing at the Ohio State University and a Consulting Associate and Director of Management Horizons, Incorporated. His specialty is the analysis of buyer behavior and development of marketing strategy.

Dr. Blackwell is the co-author of the Fourth Edition of *Consumer Behavior*, the most widely adopted textbook in the field. He has written fourteen books including *Marketing Management*, a soon to be released book relating marketing principles to corporate strategy. Some of his other books include *Consumer Attitudes Toward Health Care*, *American Attitudes Toward Death and Funerals*, *Communications for the Funeral Profession*, *Laboratory Equipment for Marketing Research*, *Research in Consumer Behavior*, and *Strategic Marketing*.

His Ph.D. is from Northwestern University and he holds B.S. and M.S. degrees from the University of Missouri. He has served as a Visiting Professor of Marketing at Stanford University Graduate School of Business and a visiting lecturer in Canada, Australia, South Africa and several European countries.

Dr. Blackwell has won several awards for outstanding teaching at the Ohio State University and he is a consultant to some of America's largest corporations. He has served as an expert witness before the Federal Trade Commission and other legal proceedings and his opinions have been quoted in the *Wall Street Journal*, *Forbes*, *Business Week* and other leading business publications. Over fifty articles he has written have appeared in *Journal of Marketing Research*, *Journal of Advertising Research*, *Business Horizons* and many other publications.

Revised 7/1/81

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The Ohio State University

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The **Chairman**. Now, you have indicated that this legislation is unnecessary because over 90 percent of our citizens now know that smoking is harmful. If this is so, it is likely due to the initial warning label required by legislation, or at least in the eyes of some people.

Now, how could this effort to inform smokers about specific health consequences be counterproductive, as you say?

Mr. **Horrigan**. Mr. Chairman, to support your concern about public health, from a marketing viewpoint, because we are really talking about delivering a message, and that is also your intent, our experience is that there is a great advantage in what we call single mindedness versus clutter. We are not permitted, as you know, to advertise on television, but manufacturers who do, worry greatly about the millions of dollars they spend on spot commercials surrounded by other commercials, which causes clutter, and therefore their message is diffused. We believe in single mindedness in effective marketing

What you in effect have here is a very effective warning that has been proven over the years, and we honestly believe that, in terms of your own sincere intentions, you would add confusion and clutter, let alone the chaos that would exist on the manufacturing side.

The **Chairman**. In your testimony, you state the Comprehensive Smoking Prevention Education Act is, "designed to force smokers to conform and to encourage prohibition of smoking." Now, of course, I think everybody here today has indicated that there is no desire to have prohibition of smoking but a desire to inform the public.

Now, I do not know whether this public education effort would be forcing or prohibiting smoking, but that is certainly not our intent.

You indicate 30 highly qualified and respected scientists were ready to appear and challenge the scientific findings of our bill. Yet, I think the overwhelming majority of scientists throughout the world accept the conclusions of thousands of researchers and their efforts linking cigarette smoking with disease and death.

Now, how do you account for the striking difference of opinion among these various scientists?

Mr. **Horrigan**. First of all, Mr. Chairman, although I have interjected myself this morning in these medical proceedings, I will not proceed to answer in a scientific way your question, but I will make this opening statement, that just prior to our appearance here, an eminent physician said that, indeed, there is a controversy that does exist, and that is a fact, after all these years.

To answer your question and to add greater perspective, I would like to turn the question over to Dr. Sommers.

The **Chairman**. Dr. Sommers?

Dr. **Sommers**. Mr. Chairman, the key word is "linking." Epidemiology, indeed, shows a strong statistical relationship between smoking and a variety of diseases, but epidemiology is a scientific technique that can never prove cause and effect.

Cause and effect have to be proved by other methods such as animal experiments and in vitro experiments, and that has not been achieved.

The **Chairman**. You suggested that this causal link between smoking and disease has not yet been established; yet all official reports from our Government for the last 14 years and at least one major World Health Organization report published in 1981 indicate loud and clear consensus.

Now, do you think that there would be any controversy if the tobacco industry did not question these studies?

Dr. **Sommers**. I repeat, sir, that I accept a statistical relationship. I do not accept a cause-and-effect relationship. I do not think anything the tobacco industry did has, is, or would affect the natural course of science except that politicization makes it difficult for science.

The **Chairman**. Let me ask you this. As I understand it, the tobacco industry has done extensive scientific research on the effects of tobacco smoking. Has that research all been made public?

Mr. **Horrigan**. You are correct, Mr. Chairman. We spend in excess of $10 million a year through no-strings-attached medical research. Those grants are awarded to eminent scientists, and the results of their research are published. I believe Dr. Sommers, who heads up the Scientific Advisory Board and directs this activity, could give some additional information.

Dr. **Sommers**. The Council for Tobacco Research, with a budget in excess of $7 million a year, accepts applications from anyone wishing to study in the field of tobacco and health. The Scientific Advisory Board considers the application. It awards the funds. If they meet the criteria of excellent science, the grants are usually for 3 years and are renewable.

There is no control exercised by the council or by the tobacco industry upon the findings. They are freely published anywhere that the investigator wants, and anything that is found is to be published.

The **Chairman**. Mr. Horrigan, you stated that the Swedish rotational warning system or scheme was not effective and that cigarette consumption has actually risen since the new system was implemented. But I have before me the latest statistics on cigarette smoking in Sweden—this comes from the Swedish Institute.

For men, the number of daily smokers has declined from 43 percent in 1976 to 31 percent in 1980. For women, the number of daily smokers has declined from 35 percent in the midseventies to 26 percent in 1980. Would you care to comment on these statistics?

Mr. **Horrigan**. I will, Mr. Chairman, because we have the same experience in the United States. The incidence rate of smoking is declining, so that when you relate the trend with regard to the incidence rate of smoking, we are experiencing that same phenomenon or development here in the United States, for both sexes.

The **Chairman**. Thank you.

Senator East?

Senator **East**. Thank you, Mr. Chairman.

Gentlemen, I wish to thank you for coming and helping us this morning on this very important matter. I would, Mr. Chairman, like to underscore what I said at the beginning of these hearings, and Mr. Horrigan has also mentioned, that I would trust we might, in order that we can give this matter the serious due that it deserves, have additional hearings so that they could be, frankly, more balanced in terms of the points of view that we need to air and look at.

I would agree with Mr. Horrigan that this question of science has loomed up large and with Dr. Sommers on the question of cause and effect as opposed to straight statistics. I do not wish to go at that ad nauseam, but it is a critical assumption that we are dealing with science here, and in American public life and in policy formulation, when the word "science" is heard, it would suggest we follow instructions, that there are no loose ends, that all has been carefully stitched together, and we must follow the dictates of science.

I do feel this morning, from my vantage point at least, that the testimony has been valuable to question whether we are talking about science or wish or judgment or value, et cetera.

Mr. Horrigan, your testimony, I think, can be particularly valuable for me and, I would hope, for the committee's record here. I had hinted at the beginning of this hearing today, and you have touched upon it, that, ultimately, those of us on this side of the issue in terms of being in the legislative chamber—and I know Senator Hatch has a great sensitivity to this and a very distinguished record in this area, as a matter of fact—of making sure that burdens placed upon industry are commensurate with some gain to the public.

The regulatory reform bill that the Senate is going to be taking up shortly hits that one head on. In the past, there has been the tendency of Government, of policy formation here in Washington, in the Senate, and in the House, frequently to dump upon businesses, or to place upon businesses, to use a more neutral phrase, regulations that just incredibly hobble an industry with no real genuine corresponding benefit to the public in terms of cost-benefit analysis or anything of this kind.

I think, obviously, you are in a very unique position because your industry would obviously bear the burden of this. I had indicated earlier, I suspect other businesses could be affected by it, too, but let's leave that for another time or place.

As you see it, this act, this particular piece of legislation, would place substantial, serious burdens upon your industry. I would like for you to comment briefly on that and just some of the illustrations of it that you see; and then, second, to weigh that as to what you see would be the gain, if any, to the public on the issue before the House, or the Senate in this case, namely the problem of smoking and public health. Where might you advise us we would be if this bill goes into operation? How would you weigh that, coming from a key figure in a key industry. Probably the R. J. Reynolds Co., which we are very proud to have in North Carolina, might very well say the industry, but we will not get into all the partisan loyalties. How would you assess that for me in general terms? It would be valuable for me, and I think it would be valuable for the record to show.

Mr. HORRIGAN. Fine, Senator. I am somewhat partisan. My son graduated from the University of Utah, so he cannot be all bad, Mr. Chairman. [Laughter.]

He is a skier and a business student.

But I think, to answer your question, Senator, we are a responsible and a concerned industry. I think we have proven that over the past. If there was substance and reason for what was being proposed here, we would be responsive to it, No. 1.

No. 2, with regard to the economic impact in terms of pure dollars and cents, we have made reference to this with regard to the House of Representatives bill. Yes, it would cost, let's say, roughly, in the range of $40 to $50 million a year, and that is a cost burden that would just continue to be passed on during what we now know to be very difficult times.

But even if you put that number aside, the fact that these statements are without foundation, without basis, along with the fact that the chaos and the confusion that would result in the marketplace for manufacturers to comply with such a law with regard to the statements I made on the number of brandstyles in the marketplace, the rotation sequence, the ability of FTC to monitor such a system; we just think that no matter how flexible we are—and we are flexible—it would be a total chaos in the marketplace, adding, of course, to the clutter and the confusion that we sincerely believe would result from a message standpoint.

Senator **East**. So the burdens on the industry are, as you see it, considerable and of deep concern to you. On a scale of difficulty of 1 to 10, perhaps, this bill causes you considerable concern, not just casual concern.

Mr. **Horrigan**. Considerable. Yes, sir.

Senator **East**. What benefit do you see to the public generally from it? I gather from what you have already said you are not sure, from an industry perspective, there will be any increased benefit if the goal, which we all seem to be agreed upon, is public awareness and information about the relative health problems of cigarette smoking.

I gather that if we put you through this bill and what the impact would be in terms of government regulation, in terms of potential suits against you—and much of this, I suppose, is unforeseen as to where it might lead—you see little, if any, real, genuine gain to the public in terms of its level of increased awareness and sensitivity to and information about the relative health hazards of smoking? Is that how you see it? In short, you said, now it is working; why place an inordinate burden upon us with no commensurate increase in terms of benefit to the public. Is that what we are saying here?

Mr. **Horrigan**. That is correct, Senator. There is nothing more dangerous in a marketing company than when a new marketing man comes in and tries to change something that is working. I would love to have a message that has the awareness level of the Surgeon General's basic warning notice.

Senator **East**. So the burden upon the industry, not only yours but all involved, the other great companies in this country involved in this business, not only would the burden be great upon you and them but the benefit to the public, at least as you people from the industry see it, would be negligible if not even counterproductive in terms of the confusion of the labels and getting away from the established pattern of a set label. The cost-benefit does not balance out, as you see it.

Mr. **Horrigan**. Precisely.

Senator **East**. I have no other questions, Mr. Chairman.

Senator **Hatch**. Thank you.

Mr. Horrigan, if these statistics are right that polled Americans are unaware, that 60 percent did not know that smoking causes most cases of bronchitis and emphysema, and even 20 percent do not know that smoking causes lung cancer and 50 percent do not know that smoking causes many heart attacks, even assuming that there are other possible health detriments to smoking, if you save just a few hundred, a few thousand or maybe 100,000 lives, it would be worth putting any labels on the cigarette packages, wouldn't it?

Mr. **Horrigan**. Mr. Chairman, I think perhaps we should address those particular numbers, and if I may, I will have Mr. Miniard answer.

Mr. **Miniard**. The problem here in looking at these numbers and interpreting that many Americans are unaware of these alleged hazards of smoking is a problem of interpretation of what they mean.

For example, most of these statements that we have before us assume that the person is willing to believe that there has been a direct causal link between smoking and a certain illness. In fact, it seems very likely that people may be aware of the relationship, or the alleged relationship, or aware of the information, but have decided that in fact it is not a causal relationship.

To give an illustration of that, one of the studies upon which I presume some of this information is based was the Chilton study in which the same people were asked two questions. One question people were asked was whether or not they agreed that heart disease was associated with smoking. For this question, only 10 percent of teenagers and the adults that were tested were considered to be uninformed.

When we moved to a more causal statement where we say smoking causes heart disease, for the same people, we find numbers ranging between 27 and 40 percent. So it seems that the problem here is that we are trying to measure knowledge in an area where we assume that a person does not possess knowledge unless they agree that smoking causes as opposed to smoking is associated with a particular health hazard.

Senator **Hatch**. I see.

Well, we thank you for coming. We appreciate your testimony, and we will keep the record open so that you can submit other testimony as well.

Mr. **Horrigan**. Thank you, Mr. Chairman and Senator East.

Senator **Hatch**. Thank you. It is nice to have you here.

With that, we will recess this hearing.

[Whereupon, at 12:31 p. m., the committee recessed, to reconvene at the call of the Chair.]

APPENDIX

ADDITIONAL STATEMENTS SUBMITTED FOR THE RECORD

American College of Cardiology
American Medical Association
American Association For Respiratory Therapy
American College of Physicians
American College of Chest Physicians
Action on Smoking and Health
Bakery, Confectionery and Tobacco Workers International Union
Domingo M. Aviado, president, Atmospheric Health Sciences, Inc.
Cigarette Smoking of Pregnant Women by Bea J. van den Berg
Peter L. Berger, professor, Boston University
Rodger L. Bick, M.D., medical director, San Joaquin Hematology & Oncology Medical Group
Theodore H. Blau, Ph. D.
Walter M. Booker, Ph. D., president, Walter M. Booker & Associates, Inc.
Oliver Gilbert Brooke, M.D., FRCP, Department of Child Health, St. George's Hospital, London
Barbara B. Brown, Ph. D.
Victor Buhler, M.D., pathologist, Kansas City, Mo.
Jack Matthews, M.D., Emeritus Professor of Surgery, University of California at San Diego
Sherwin J. Feinhandler, Ph. D., president, Social Systems Analysts, Inc.
Edwin R. Fisher, M.D., professor of pathology, University of Pittsburgh School of Medicine
H. Russell Fisher, M.D., Emeritus Professor of Pathology, University of Southern California
Jean Dickinson Gibbons, professor of statistics, University of Alabama
Katherine McDermott Herrold, certified pathologist
Arthur Furst, Ph. D., Institute of Chemical Biology, University of San Francisco
Richard J. Hickey, Ph. D., senior research investigator, department of statistics, Wharton School, University of Pennsylvania
Duncan Hutcheon, M.D., D. Phil., departments of pharmacology and medicience, New Jersey Medical School
Leon O. Jacobson, University of Chicago
Lawrence L. Kupper, professor of biostatistics, School Of Public Health, University of North Carolina at Chapel Hill
Hiram Thomas Langston, M.D., clinical professor of surgery (emeritus), Northwestern University Medical School
S. C. Littlechild, B. Com. Ph. D., professor of commerce, University of Birmingham
Eleanor J. Macdonald, professor emeritus, department of cancer prevention, University of Texas System Cancer Center, Houston, Tex.
John E. O'Toole, chairman, Foote, Cone & Belding Communications, Inc.
L. G. S. S. Rao, Ph. D., Bellshill Maternity Hospital, Bellshill, Scotland, U.K.
Dr. Jay Roberts, professor and chairman of the department of pharmacology, Medical College of Pennsylvania, Philadelphia
Henry Rothchild, M.D., Ph. D., professor of medicine and anatomy, Louisiana State University
Dr. Henry I. Russek, practising specialist in the filed of cardiovascular disease
Bernice C. Sachs, M.D., Seattle, Wash.
G. N. Schrauzer, Ph. D., La Jolla, Calif.
Dr. Carl C. Seltzer
Sheldon C. Sommers, M.D.
Theodore Sterling, University Research Professor at Simon Fraser University, Burnaby British Columbia
Yoram J. Wind, professor of marketing, Wharton School, University of Pennsylvania
Tobacco Institute, theRobert Casad Hockett, research director, Council for Tobacco Research--U.S.A., Inc.
Charles D. Spielberger, Ph. D., University of South Florida, Tampa, Fla.

American College of Cardiology

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STATEMENT

on

S. 1929

"Comprehensive Smoking Prevention

Education Act of 1981"

of

Dan G. McNamara, M.D., F.A.C.C.
President

to

Honorable Orrin G. Hatch

Chairman
Committee on Labor and Human Resources

March 16, 1982

Dear Mr. Chairman and
Members of the Committee:

I am Dan G. McNamara, M.D., F.A.C.C., Professor of Pediatrics and Chief of the Cardiology Section, Baylor College of Medicine and Texas Children's Hospital. I am also President of the American College of Cardiology, a professional medical specialty society representing over 11,500 physicians, scientists and educators who specialize in diseases of the heart and circulatory system. ACC is dedicated to ensuring optimal care for persons with cardiovascular disease and those with the potential for developing cardiovascular disease and, through educational and socioeconomic activities, to contribute significantly to the prevention of cardiovascular disease. It is in my capacity as President of the College that this statement is made on S. 1929, the "Comprehensive Smoking Prevention Education Act of 1981."

One main concern of the College is whether consumers receive sufficient and accurate information on the demonstrated relationships between cigarette smoking and cardiovascular disease. The College believes that consumers are provided with insufficient information on the demonstrated relationships between cigarette smoking and cardiovascular disease and that this lack of information weakens a physician's role in altering a patient's smoking behavior.

As the Federal Trade Commission noted in its "Staff Report on the Cigarette Advertising Investigation" (May, 1981), which we commented on (attached), a "substantial portion of the public remains uninformed about the hazards of smoking," and that FTC tests "indicate that the (current) warning is neither noticed nor read by the vast majority of people." Even though the current warning indicates that the Surgeon General has determined that cigarette smoking is dangerous to one's health, 17% of all smokers and 24% of heavy smokers do not know that smoking is hazardous to health. Additionally, as noted, consumers themselves agree that the current warning is inadequate.

Why not provide the public with the type and quality of information sought? As the FTC report notes, nearly "two out of every three questioned during the 1978 Roper survey for the Tobacco Institute, believe that the current warning is not "adequate" and indicated they preferred a health warning that describes the specific health risks of smoking."

A 1978 Gallup survey of smokers and nonsmokers found that 32% were not aware of the statistical relationship between cigarette smoking and heart attack; that 37% of smokers did not know this relationship; and that among heavier smokers, 40% did not know the increased risk for heart attack associated with cigarette smoking. The 1979 Chilton Study, conducted by the FTC, found that 25% of the general population and 31% of smokers did not know that smoking greatly increased their risk of heart attack despite research that concluded that it doubles a person's risk of heart attack. In addition, consumer knowledge of the relationship among cigarette smoking, birth control pills and heart attack is low, despite the fact that women who smoke *and* take birth control pills have approximately *ten* times the risk of a heart attack of women who do neither.

Therefore, the College agrees that the current cigarette warning is not effective relative to the public's need to know and act on the strong scientifically-validated statistical relationship between cigarette smoking and cardiovascular disease. The College agrees with the need to improve on the type and content of the required cigarette warning labels.

Therefore, in reviewing the FTC report and its conclusions, the College endorses the concept of rotational warnings as being more effective than the current warning.

The College believes that the labeling approach and specific warnings contained in S. 1929 (as modified below for scientific acceptability) will assist consumers in making conscious, more informed decisions about smoking, because they will have more definitive information on the specific health consequences of smoking. This type of information, also,will assist cardiologists, other physicians and other health professionals in communicating the risks of smoking to their patients.

The College would recommend that "WARNING: The Surgeon General has Determined that Cigarette Smoking Causes Heart Disease" be amended as follows: "Current scientific evidence indicates that Cigarette Smoking is a Major Risk Factor for Coronary Heart Disease." The College would also recommend that "Current scientific evidence indicates that cigarette smokers are predisposed to sudden death," and "Current scientific evidence indicates that there is a significant relationship between cigarette smoking and heart disease, cancer or emphysema" be considered.

In addition, the College believes that one aspect of the wording of the bill relating to the coordination of research, conduct of research and the authority of the Secretary to carry out the provisions of this bill through grants, may need clarification to avoid duplication with research on cigarette smoking effects being conducted by NHLBI, NCI and other Institutes at NIH. If coordination means the implementation of a systematic exchange of information on the research being supported by different agencies, this would be useful. If, however, the purpose is to provide the Office of Smoking and Health with authority to direct the research of Institutes, the College believes it would be counter-productive and undesirable.

However, authority to conduct types of research that do not fall clearly under the authority of the categorical Institutes could be appropriate for the Office of Smoking and Health. Examples of such would be in monitoring national trends of cigarette consumption in different age groups, surveillance of the composition of tar, nicotine, carbon monoxide and other potentially harmful constituents of cigarettes and maintaining the periodic reporting of new research findings.

In addition, the College also believes that the Congress should review all the current material on the connection between cigarette smoking and the costs to society through increased medical costs and reduced productivity in order to provide a basis for further cigarette smoking prevention efforts.

I trust that these comments that relate to our support for your efforts in this area will be of assistance to you and the Committee. We would be pleased to provide you with any other assistance.

American College of Cardiology

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December 10, 1981

James C. Miller, III
Chairman
Federal Trade Commission
Sixth Street and Pennsylvania Avenue, N.W.
Washington, D.C. 20580

Re: File Number 792-3204

Dear Mr. Chairman:

The American College of Cardiology, a professional medical specialty society representing over 11,000 physicians, scientists and educators who specialize in diseases of the heart and circulatory system, is pleased to provide initial comments, with respect to the Staff Report on the Cigarette Advertising Investigation of the Federal Trade Commission, May 1981.

The College believes the FTC has provided a useful and constructive contribution to the health hazards of cigarette smoking. Enclosed is the Bethesda Conference Report on the "Prevention of Coronary Heart Disease", which the College hopes you will find instructive on our commitment to prevention in such areas as cigarette smoking. The conference, held September 27-28, 1980 in Bethesda Maryland at the American College of Cardiology, focused on the potential for physicians to do much more in the coronary heart disease prevention effort, especially in concert with allied health professionals The College endorses the recommendations of this conference report.

I would like to note that the mission of the American College of Cardiology is to ensure optimal care for persons with cardiovascular disease or the potential for developing it; and ultimately, through appropriate educational and socioeconomic activities, to contribute significantly to the prevention of cardiovascular disease.

The Bethesda Conference Report noted that cigarette smoking is one of the major coronary risk factors. Page 26 details concerns highlighted in the FTC Report concerning the competition between the dangers of cigarette smoking and the attractive modeling and glamor of cigarette smoking.

The Bethesda Conference Report embodies many of the concerns enumerated in the FTC Report, specifically the need to have credible information provided in a manner which will be believed and which will provide either positive behavior modification or prevent initial negative behavior.

The College believes that there has not been sufficient time permitted for us to provide the breadth of input from the various components of the College, particularly the Preventive Cardiovascular Disease Committee, on these recommendations to allow us to more fully address this issue, which we consider to be of paramount importance. Please be assured that our additional views will be provided to you as soon as possible. In addition, we will share with the FTC our comments on current and proposed legislation on this subject. We trust you will find these future comments of assistance.

Please let me or Roger C. Courtney, J.D., ACC Director of Government Relations, know if you need any other assistance on this or any other matter.

Sincerely,

Dan G. McNamara

Dan G. McNamara, M.D., F.A.C.C.
President

DGM:cs

Enclosure

STATEMENT

of the

AMERICAN MEDICAL ASSOCIATION

to the

Labor and Human Resources Committee

U.S. Senate

Re: S. 1929 - Comprehensive Smoking Prevention Education Act

March 16, 1982

The American Medical Association takes this opportunity to comment on S. 1929. The bill states its purpose is "to provide a new strategy" to educate and provide information to the American public that will allow individuals to make informed decisions concerning smoking." The bill would accomplish this by replacing the current general health warning found on cigarette packages with seven specific health warnings. One of the seven specific health warnings would be required on all cigarette packages and in advertisements. The warnings would be rotated among brands so that each brand would use all warnings within a fifteen-month period. S. 1929 would also require the "tar," nicotine and carbon monoxide levels to be disclosed on packages and advertisements. Cigarette manufacturers would be required to provide the Federal Trade

Commission with a list of the chemical additives in each of their brands of cigarettes. This information would be protected as a trade secret and used only for research purposes.

Comments

The United States Surgeon General stated in his recent report, "The Health Consequences of Smoking," that "Cigarette smoking . . . is the chief, single, avoidable cause of death in our society and the most important public health issue of our time."

A decision to smoke should be made with the knowledge that increased health risks are associated with smoking. For this reason the AMA is supportive of efforts to increase public awareness of the hazards.

The AMA has been involved in many efforts to increase the public's knowledge of the consequences of smoking. The AMA receives and answers many requests for smoking information. In 1978 the AMA published "Tobacco and Health," an account of the comprehensive research program conducted by the AMA. A pamphlet called "Smoking: Facts You Should Know," a copy of which is attached, has been widely distributed along with two anti-smoking posters. Physicians are urged to alert smokers to the risks associated with smoking.

Our comments address only the rotational labeling provisions. We find it very disturbing if, as a recent FTC staff report alleges, approximately ten percent of the population still do not know that cigarette smoking is harmful. Believing a more detailed warning would better inform the public of the harm of smoking, the AMA House of Delegates adopted a report encouraging Congress to require a more explicit warning on cigarette packages.

In testifying before the Subcommittee on Health and Scientific Research of the Senate Committee on Human Resources in 1978 on a similar labeling provision contained in S. 3115, (95th Congress) the Disease Prevention and Health Promotion Act of 1978, the AMA endorsed the rotational label warning concept. We recommended at that time that if adopted the rotational labels "should be evaluated after a period of use to see if there has been any substantial difference in the public's awareness of health problems associated with cigarette smoking." We still believe the effectiveness of the labels should be evaluated.

A more explicit warning, while an improvement, will not be a complete solution. The 1980 Surgeon General's Report on Smoking and Women reveals that smoking is increasing among teenagers. The AMA is concerned about teenage smoking and does not believe a change in the warning alone will fully deal with the problem. Educational programs that emphasize the harmful aspects of smoking from a teenager's point of view should be pursued, and the use of "role models" in cigarette advertisements should be eliminated. Issues of concern to an adult, like a decrease in life expectancy, may not influence a teenager's decision to smoke. More information is needed on youth smoking, such as why they begin to smoke, why they quit smoking, and what method is most effective in urging teenagers to quit smoking. Once this information is available efforts to reduce teenage smoking can be better directed.

Even those who are aware of the dangers of smoking may have difficulty quitting because of the addictive qualities of cigarettes. The recent Surgeon General's report shows that up to 50 percent of those who quit smoking on their own will stay off cigarettes. Assistance must be provided to the remaining 50 percent if smoking is to be decreased significantly. The AMA is developing an audiovisual presentation on how to quit smoking that will soon be available for physicians to use in assisting patients desiring to quit smoking. If adopted, a change in the warning may create more awareness of the dangers of smoking, and more smokers will want to stop smoking. Programs to assist those who want to quit smoking will be needed even more in that case.

Conclusion

The AMA supports efforts to increase public awareness of the hazards of smoking. We believe that the best method to decrease smoking is to help people avoid starting the habit. One method to discourage smoking would be a clear indication of the health hazards of smoking. Thus, the AMA supports a more explicit warning on cigarette packages and in advertisements.

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AART AMERICAN ASSOCIATION FOR RESPIRATORY THERAPY 1720 Regal Row, P.O. Box 35287 Dallas, TX 75235 (214) 630-3540

STATEMENT ON S. 1929

the

COMPREHENSIVE SMOKING PREVENTION EDUCATION ACT OF 1981

by

JOHN R. WALTON, RRT

PRESIDENT

Sponsored by the American College of Chest Physicians, the American Society of Anesthesiologists, and the American Thoracic Society
Member of the National Health Council

As President of the American Association for Respiratory Therapy (AART), I welcome the opportunity to submit a statement for the record of S. 1929, the "Comprehensive Smoking Prevention Education Act of 1981". The AART is a 23,000 member professional health care organization representing respiratory therapy practitioners across the nation. Respiratory therapy practitioners deliver care to patients suffering from both acute and chronic respiratory diseases. For example, asthma, emphysema, bronchitis, black and brown lung diseases, are among the most common conditions that are treated by respiratory therapy. These conditions are aggravated and exacerbated by cigarette smoking. The respiratory therapy community has a great concern for the detrimental effects of cigarette smoking on the health of their patients. As an Association we are also concerned with an aspect of the smoking issue often neglected by other organizations, that is the hazards of passive smoking.

Passive smoking can be defined as the smoke emitted from the end of a cigarette, cigar, or pipe (side-stream smoke), and the smoke exhaled by the smoker (mainstream smoke) which comes into contact with others.

Tobacco is a complex mixture of particles, liquids, and gases, which combine into a variety of toxic and hazardous compounds when burned. Compounds such as tar, nicotine, carbon monoxide, cadmium, nitrogen dioxide, ammonia, benzene, formaldehyde, hydrogen sulphide, are among the most dangerous to the individual whether they are non-smokers or smokers.

Non-smokers need to be aware of the disturbing fact that side-stream smoke has higher concentrations of noxious compounds than mainstream smoke inhaled by the smoker. For example, side-stream smoke can have twice as much tar and nicotine, three times as much 3-4 benzpyrene, five times as much carbon monoxide, and fifty times as much ammonia as mainstream smoke does.

Perhaps one of the most hazardous compounds emitted by a burning cigarette is carbon monoxide. This colorless, ordorless gas is created by incomplete combustion, and both car exhaust and tobacco smoke put it into the air. When inhaled the carbon monoxide gas "bumps" oxygen molecules out of the redblood cells, preventing oxygen from attaching to the cell and forming a new compound called carboxyhemoglobin. Carbon monoxide also remains in the bloodstream and is difficult to remove. After three or four hours, half of the excess carbon monoxide is still in the bloodstream.

A study done in 1980 by James R. White, Ph.D. and Herman F. Froeb, M.D. evaluated the long term effects of passive and voluntary smoking on the small airway functions in 2100 middle aged subjects.[1] The findings showed that regardless of sex, non-smokers chronically exposed to tobacco smoke have lower flow rates of exhaled air than non-smokers not exposed. In addition, values in passive smokers were not significantly different from those in light smokers and those who did not inhale.

One of the most recent and most shocking research studies has been published in Japan. Conducted Takeshi Hirayama, M.D. of Tokyo's National Cancer Center Research Institute, 91,540 non-smoking wives aged 40 years and above were followed for 14 years.[2] The study showed that non-smoking women married to heavy smoking men (20 or more cigarettes a day) were up to four times as likely to die of lung cancer as women married to non-smokers.

Other studies have shown that:

1. Children with a smoking parent or parents have nearly twice as many respiratory illnesses as compared to those children of non-smoking parents.
2. Exposure to passive smoke can hasten the onset of angina in people with heart disease.

3. Individuals suffering from asthma can severely aggravate their condition by exposure to cigarette smoke.
4. It can damage the action of cilia, the microscopic hairs that line the airways of the lungs.
5. In healthy, nonallergic people it has been shown to cause eye irritation, sneezing, coughing, sore throat, hoarsenss, headache, and in some cases more serious side effects.

As more research is conducted and the results tabulated the evidence on the negative effects of passive smoking mount. It has already been shown to be extremely toxic, harmful, and carcinogenic. The public must be made aware of the harmful effects of this habit. The bill S.1929, makes great progress in educating the public on the specific dangers of smoking. We urge the Senate Committees, when amending this bill, not to neglect the serious and often overlooked are of the passive smoker.

In addition, the Office on Smoking and Health must recieve adequate and steadfast government commitment relevant to this entire issue. Committee members and government officials must not allow political considerations and pressures from the tobacco industry to negatively sway or change their opinions on an issue and product that contributes to over 300,000 premature deaths and over 25 billion dollars in lost productivity each year.

Research and public education must continue and increase markedly if we are to have any hope of reducing the tremendous social expense, irritation, suffering and death from the most preventable cause - cigarette smoking.

REFERENCES

1. White, J.S., Froeb, H.F., "Small Airways Dysfunction in Non-smokers Chronically Exposed to Tobacco Smoke", New England Journal of Medicine, 302: 1980, 720-723.
2. Hirayama, T., "Non-smoking Wives of Heavy Smokers Have a Higher Risk of Lung Cancer", British Medical Journal, 282: Jan. 17, 1981.

STATEMENT
OF
THE AMERICAN COLLEGE OF PHYSICIANS
on
S. 1929, the "Comprehensive Smoking Prevention Education Act of 1981"

Mr. Chairman and Members of the Committee:

The American College of Physicians (ACP) is pleased to have this opportunity to provide its comments on S. 1929, the "Comprehensive Smoking Prevention Education Act of 1981."

As you know, the College represents over 54,000 doctors of internal medicine, related non-surgical specialists, and physicians-in-training. The ACP membership includes private practitioners delivering primary health care; medical specialists in such fields as gastroenterology, endocrinology, oncology, and cardiology; medical educators; and researchers.

The legislation presently before the Committee seeks to establish a new strategy for educating and providing information to the American public about the hazards of smoking. It would thereby allow members of the public to make more fully informed decisions as to whether they will choose to smoke. The American College of Physicians strongly supports the central purpose of the legislation and believes that such an effort represents an important and appropriate governmental initiative -- an initiative which we believe is clearly in the public health interest of this Nation.

We concur in the scientific finding that cigarette smoking is the single most important preventable cause of illness and premature death in the United States. In particular, the American College of Physicians believes that the most recent report of the Surgeon General, "The Health Consequences of Smoking," both warrants and requires the full attention of health professionals and policy makers concerned with the protection of the public health. We believe the statements that "cigarette smoking is the major single cause of cancer mortality in the United States" and that there "is no single action an individual can take to reduce the risk of cancer more effectively than quitting smoking" lend strong suport to this Committee's efforts to more fully inform the public of the health risks attendant to smoking.

Despite statements of some to the contrary, there is no scientific dispute with regard to the numerous health hazards presented by smoking. Cigarette smoking is a major cause of cancers of the lung, larynx, esophagus, and oral cavity. Smoking is a contributory factor in cancers of the urinary bladder, kidney, and pancreas. It is a major cause of chronic bronchitis and emphysema and a major risk factor for coronary heart disease and arteriosclerotic peripheral vascular disease. Estimated deaths related to smoking are in excess of 300,000 annually. Lung cancer alone accounts for one out of four of all cancer deaths and it is estimated that at least thirty percent of all cancer deaths are attributable to tobacco use.

It is important to emphasize that at this time, despite the advances made in the treatment of certain cancers, the five year survival rate for lung cancer is less than ten percent. Despite advances in our ability to perform early diagnosis and treatment we have not significantly altered this survival rate. The best preventive measure with regard to cancer of the lung is not to smoke -- this means that those who smoke should stop, and that those who do not smoke should not start.

Lastly, important evidence has been accumulated on the effect of maternal smoking on pregnancy outcomes and the well-being of infants, and there is emerging evidence on the adverse impact of smoking on healthy non-smokers. This latter evidence challenges any statement that smoking is solely a matter of individual choice.

The fact that new evidence is being developed on the impact of smoking on health must not diminish the fact that there already exists a substantial body of irrefutable scientific evidence with regard to the health hazards and increased premature mortality associated with smoking. It should be emphasized that there is clear scientific consensus as to the veracity and import of this existing evidence.

In view of the cost to society -- in both human and economic terms -- which smoking represents and in view of the recognized need to be more diligent as a society in our efforts at health promotion and disease prevention, the American College of Physicians believes that every reasonable effort should be made to alert the public to the range and magnitude of the risks associated with smoking. It is imperative that we make every effort to ensure that those who may choose to smoke are fully cognizant of the risks to their health and the health of others which are being incurred by this action. In addition, we would note that the emerging scientific evidence with regard to the addictive properties of tobacco smoking raises new and additional concerns, and that efforts must be made to convey this information to the American public.

As physicians, in particular as specialists in internal medicine and its related subspecialties, we are fully aware of the overwhelming scientific support for the range of specific warnings outlined in the legislation before the Committee. As providers of primary and continuing care to adults, we have a strong interest in preventive health measures and in the mechanisms by which health-related information is conveyed. We support the proposed changes in the present labeling requirements for cigarette packages. As we have stated, given the clear and dramatic health risks associated with smoking, we believe that every reasonable effort should be made to accurately convey to individuals the nature of this health risk.

We believe that the specificity of the warnings outlined in the proposed legislation will help to emphasize the risk which individuals take every time they smoke. We also believe that it is vitally important to convey to smokers the fact that quitting smoking will reduce the risks to their health. We are pleased to see that under the proposed legislation both messages would be presented. Some might argue that the information conveyed by the proposed warnings is too graphic, but we would counter that the scientific evidence underlying these warnings is also quite graphic. We believe that the specificity of the warnings and their prominence on the packaging materials will provide an important adjunct to our efforts as health professionals to counsel patients in maintaining sound health and reducing the risk of preventable disease and illnesss.

Some have argued that there is not sufficient evidence of the "effectiveness" of the proposed system of rotational health warnings. However, we believe that such an argument is without merit in the face of such substantial and clearcut evidence of adverse health effects and premature mortality, and in view of the tremendous social costs associated with smoking. It is our belief that this preponderance of evidence carries with it a responsibility and in fact a duty to warn the public regardless of whether statistics are presently available which indicate that the public will in fact heed this warning.

We also believe that in the interests of sound international health policy -- and in keeping with a longstanding commitment to being cognizant of the health needs of all people -- warnings should appear on all cigarette packages produced and manufactured in this country, irrespective of where they are ultimately offered for sale.

In addition, we support the provisions included in the proposed legislation which would require that nicotine, tar, and carbon monoxide levels be disclosed. We also support efforts to require that information be provided to the Federal Trade Commission and the Department of Health and Human Services with regard to those chemical additives used in the manufacture of cigarettes and the quantities of such additives.

At the present time it is extremely difficult to fully assess the relative risks of cigarette additives because of this lack of basic information. Those listings of additives which presently exist are not sufficiently specific in terms of the types and quantities of substances actually being used. The presently unknown health effects of cigarette additives must be explored and such an assessment can only be undertaken if the scientific community has access to more complete information. Given our growing national concern with toxic substances and our developing knowledge with regard to the possible synergistic effects of certain substances, we believe that a national compendium of information on cigarette additives should be considered if we expect to be able to assess present and future health risks.

In conclusion, Mr. Chairman, we support efforts to better inform the public of the health hazards, including the increased risk of premature mortality, associated with smoking. As public policy turns increasingly towards emphasizing the importance of individual efforts at disease prevention and health maintenance we believe that it is essential that individuals receive complete information on the risks associated with smoking. It is our belief that the measure before this Committee will substantially enhance our efforts as health professionals to convey vitally important scientific information to the public.

The American College of Physicians is pleased to lend its support to your efforts and is available to respond to any questions which you might have.

Testimony of

THE AMERICAN COLLEGE OF CHEST PHYSICIANS

Submitted by

Thomas L. Petty, M.D., F.C.C.P.
President

regarding

S. 1929: "The Comprehensive Smoking
Prevention Education Act of 1982"

Senate Labor and Human Resources Committee
Senator Orrin G. Hatch, Chairman

March 16, 1982

Senator Hatch and Members of the Committee:

I am Dr. Thomas Petty, Professor of Medicine and Anesthesiology, Head of Division of Pulmonary Sciences, University of Colorado Health Sciences Center. I am also the President of the American College of Chest Physicians, a professional medical specialty society of more than 11,000 physicians, scientists, and educators who specialize in diseases of the heart, lungs, and circulatory system. It is in this latter capacity that I express our support for S.1929, "The Comprehensive Smoking Prevention Education Act of 1982."

As pulmonary and cardiology specialists, we have seen first-hand the significant health problems associated with smoking and recognize that we are in a unique position to influence our patients to forego smoking in the first place and to stop smoking when health conditions so require. Accordingly, as a society committed to post-graduate medical education, we have viewed the education of physicians (who in turn educate their patients) on the smoking problem as one of our highest priorities.

-- In 1968, the College supported in conjunction with the National Clearing House for Smoking and Health, a national forum on office management of smoking. The procedures of this conference were published in CHEST, the official journal of ACCP.

-- ACCP joined the National Cancer Institute in the preparation of a correspondence course on smoking and its relationship to chronic obstructive lung diseases and asbestosis. This course was mailed to tens of thousands of primary care physicians.

-- The national media focused on our 1972 Annual Scientific Assembly, when the College established a policy requiring smoking physician-registrants to sit in a special smoking section in the assembly room. This section is no longer utilized because so few of our national convention participants now smoke. Since 1980, new Fellows of the College have been asked to take a pledge that they will use their offices, clinics, and hospital environments as health centers to discourage smoking.

-- The College is preparing for publication in CHEST a document describing procedures that the physician should utilize in the office, clinic, and hospital to discourage smoking and to identify the pulmonary specialist's role in smoking cessation.

-- The College is preparing a monograph for distribution in October 1982 identifying the responsibility of chest physicians to inquire of all their patients about their smoking habits and to assist patients in smoking cessation.

The impact of cigarette-smoking is well documented. It causes approximately 300,000 deaths annually; this is one out of seven of all deaths in the United States. In 1980 over 80,000 individuals died from smoking-related emphysema and chronic bronchitis; and approximately 200,000 heart attack deaths were attributed to smoking. The Surgeon General's latest report concludes that "cigarette smokers have overall mortality rates substantially greater than those of the non-smokers."

The statistics on the impact of cigarette-smoking are staggering and clearly call for relief in the form of the proposed legislation. Regarding lung cancer, the Surgeon General's report states that:

1. Cigarette smoking is the major cause of lung cancer in the United States.

2. Smokers who consume two or more packs per day have lung cancer mortality rates 15-25 times greater than non-smokers.

3. Cessation of smoking reduces the risk of lung cancer mortality compared to that of the continuant smoker.

4. The economic impact of lung cancer to the U.S. is approximately $4.25 billion per year in lost earnings, hospital costs, and physician fees.

5. Lung cancer is a preventable disease. It is estimated that 85% of lung cancer mortalities could be avoided if individuals had never taken up smoking.

Over one-third of our membership are cardiologists; they see in their offices and clinics the devastating implications of smoking vis-a-vis cardiovascular diseases.

1. Smoking is a major contributor to the occurrence of heart attacks, sudden death and peripheral vascular disease.

2. Smoking doubles a persons' risk of heart attack and heavy smokers are three times as likely to suffer heart attacks and sudden death.

3. Cessation from smoking greatly reduces the risk of cardiovascular disease.

4. The economic costs of cardiovascular disease to the U.S. is approximately $41.8 billion per year in lost earnings

and health costs, both Federal and private.

Other chronic respiratory diseases are also caused or aggravated by the direct inhalation of cigarette smoke, including chronic bronchitis, asthma, emphysema, pleurisy, pneumoconiosis, bronchiectasis and other disorders. Over 16 million Americans suffer from these diseases.

The most salient characteristic of the smoking problem is that not smoking or ceasing smoking is a means of preventing or delaying the onset of the above-mentioned lung and cardiovascular disorders. This Committee has been apprised of numerous studies that demonstrate increased life expectancy in individuals who stop smoking as compared to those who continued to do so. A twenty-year study(British Medical Journal, 2: 1525-1536, 1976) of over 34,000 physicians in Great Britain showed that deaths from chronic obstructive lung diseases decreased by 24% for those who stopped smoking. The death rate from lung cancer for those who continued to smoke was 16 times higher than non-smokers; after 15 years of abstinence by smokers their death rate from lung cancer fell to only twice the rate of non-smokers. Most recently, an American Study (American Review of Respiratory Diseases, 125:144-51, 1982) documents that cigarette smoking in adults is the most important predictor of obstructive airways disease and its cessation significantly reduces the future development of the diseases. The use of a simple spirogram (pulmonary function test) markedly enhances the ability to predict obstructive airways diseases.

Another study (New England Journal of Medicine, 300: 213-217, 1979), showed that individuals with coronary artery the claims of the tobacco industry that there does not yet exist a scientific causal link between cigarette smoking and major cardio-pulmonary health problems, the evidence is quite clear. Government intervention in the marketplace is desirable when the problem is one of national scope, when the costs of regulation are clearly outweighed by benefit to society, and when the government can perform a function not assumable by the private sector. A visible governmental commitment -- or recommitment -- to the eradication of smoking is clearly required. We believe that the passage of S. 1929 will do much to assist we practitioners in educating the public regarding health risks involved with smoking cigarettes. We would like to focus on each of the major provisions contained in this legislation.

1. We support the establishment of a program to inform the public of the dangers to health from cigarette smoking. The voluntary sector cannot bear the entire responsibility for educating the public on the risks of smoking. The high cost of advertising and the volume and influence of cigarette advertising are major deterrents to voluntary efforts. The establishment of an Interagency Committee on Smoking and Health to coordinate research and educational efforts of the Federal Government and private sectors will help to avoid duplication of research already being conducted by NIH or other Federal agencies. It is important that the NHLBI and NCI, as well as the other Federal agencies you have mentioned, be represented on the Interagency Committee.

We believe that the bill should be amended to establish by statute the Office of Smoking and Health within HHS so that there may be a permanent focus for Federal activities in this area. Smoking and its health problems are too significant to be subject to the economic or political climate. The Office of Management and Budget, in its effort to reduce domestic spending, has tried to zero-fund the Office twice. Thanks to the efforts of Secretary Schweiker, the Office has retained its strength and integrity. A Congressional mandate for the Office will assure continuing public education on the hazards of smoking, and will demonstrate Congress' commitment to a healthier America.

2. We also support the provision that would require the rotation of five new warning labels to appear on cigarette packages and in advertising. The Federal Trade Commission recently determined that "current cigarette advertising practices may mislead consumers by omitting material facts about the health risks of smoking". The report indicated that consumers do not know enough about the harmful effects of smoking and often underestimate the risks of suffering from health problems related to smoking. The current warning label is overexposed and too non-specific as to the health hazards of smoking. We feel that disease-specific warning labels tailored to varying populations (such as pregnant women) will more effectively inform consumers about the health problems associated with smoking. Rotating the labels so that the label statements will appear an equal number of times on each brand of cigarettes and advertising during a fifteen month

period will aid in efforts to get more up-to-date information to the public in the future. Varied warnings will also promote greater dialogue between physicians and their patients regarding smoking.

3. We strongly concur that advertising information and cigarette packages should be required to identify the carbon monoxide yield of cigarette smoke as well as the content of tar and nicotine. Carbon monoxide is one of the most harmful ingredients of cigarette smoke, in that it reduces the blood's ability to carry oxygen to the cells. Carbon monoxide is particularly harmful to pregnant women and may also be a critical factor in coronary heart disease, sudden death, atherosclerosis, and chronic respiratory diseases.

4. S. 1929 should be amended to conform to provisions in the companion House legislation (H.R. 5653) which increase the civil penalties for violation of the new labelling requirements outlined in the bill.

In conclusion, we feel that the evidence of the need for a national education effort on the hazards of smoking is overwhelming. Study after study has established the link between smoking and lung cancer, cardiovascular diseases and many other disorders. Consumers do not have all of the facts to make an educated choice between not smoking and smoking. Cigarette manufacturers spend $1.0 billion on advertising their products each year, or 50 times the amount expended on public education The statutory establishment of an Office on Smoking and Health will insure a continuing national education effort on the dangers of smoking and rotational labelling will maximize exposure of millions of Americans to at least a minimum level of information on smoking's health hazards.

We heartily welcome the expanded Federal role in addressing the smoking menace to this Nation's health. Mr. Chairman, you and your colleagues are to be commended for recognizing that this problem is one of national scope and deserving of a national legislative and administrative focus. We, as practitioners, educators, and researchers, are pleased to have the opportunity to join hands with the Federal Government to help eradicate the human, social, and economic waste resulting from smoking.

ASH
ACTION ON SMOKING AND HEALTH
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TESTIMONY OF ACTION ON SMOKING AND HEALTH (ASH), BY ITS EXECUTIVE
DIRECTOR AND CHIEF COUNSEL, JOHN F. BANZHAF III, BEFORE THE
SENATE COMMITTEE ON LABOR AND HUMAN RESOURCES, CHAIRED BY
THE HONORABLE ORRIN G. HATCH, ON THE COMPREHENSIVE
SMOKING PREVENTION EDUCATION ACT (S. 1929)
Submitted April 2, 1982

I am happy to appear before you on behalf of Action on Smoking and Health (ASH) to strongly support The Comprehensive Smoking Prevention Education Act. As you may know, Action on Smoking and Health is a national nonprofit organization which serves as the legal action arm of the antismoking community. In this capacity it has been directly involved in virtually all major actions concerning cigarette smoking and advertising.

For example, in my individual capacity I filed the complaint at the Federal Communications Commission which led to the requirement that stations make free time available for antismoking messages under the Fairness Doctrine. Shortly thereafter ASH was formed and played a major role in upholding the Congressionally-imposed ban on cigarette advertising. ASH was also instrumental in forcing "little cigar" ads off the air and in persuading the Federal Trade Commission to file complaints against the tobacco industry concerning various cigarette advertisements.

In my individual capacity as a Professor of Law at the National Law Center of the George Washington University I was able to persuade the Federal Trade Commission to adopt corrective advertising as a weapon against deceptive ads. Since that time I have participated in numerous proceedings concerning deceptive advertising at the Commission, including a major fact-finding proceeding concerning the permanent effects of advertising.

On the basis of this experience it is my judgment that the requirement of stronger, clearer, more specific health warnings is a necessary and long-overdue step. This, coupled with the idea of changing, or "rotating," warnings will do much to make them more effective and to bring them forcefully to the public's attention.

Because the need for these warnings has been amply demonstrated in other testimony and in many studies by the Federal Trade Commission and other agencies, I will not dwell on this aspect. However, I think it is important for the Congress to appreciate the depths to which the tobacco industry will descend to try to lure young people into smoking and to negate or otherwise distract their attention from the serious health problems it presents. To dramatize this, I am attaching, as part of my testimony, a portion of a confidential report prepared by the Staff of the Federal Trade Commission and -- for reasons one may well conjecture -- not made available to Congress or the public.

The report demonstrates many things. First, the industry apparently has nothing but contempt for people who use its product or people who might be persuaded to do so. For example, a tobacco industry report at page 2-16 of the Federal Trade Commission's confidential document says,

> Thus, the smokers have to face the fact that they are illogical, irrational and stupid . . .
>
> * * * *
>
> . . . smokers don't like to be reminded of the fact that they are illogical and irrational.

The report also demonstrates that the tobacco industry actively encourages smoking by young people and actively designs campaigns to encourage them to begin smoking. For example, among the strategies for attracting young "starters" to cigarette smoking are the following:

> Present the cigarette as one of a few initiations into the adult world.
>
> Present the cigarette as part of the illicit pleasure category of products and activities.
>
> * * * *
>
> To the best of your ability, (considering some legal constraints), relate the cigarette to 'pot', wine, beer, sex, etc.

Third, this confidential document makes it abundantly clear that the tobacco companies use a variety of means to detract from and undermine the health warning on cigarettes. On this point I think the report speaks most forcefully for itself.

In addition to including this report as a part of ASH's testimony, Action on Smoking and Health would like to respectfully request and suggest that the Committee demand from the Federal Trade Commission the various documents cited in the report and place them, also, in the record, so that Members of Congress may be fully advised on the tobacco industry's strategy and techniques and the effectiveness of their advertisements prior to a vote on this issue.

RECEIVED MAR 1 5 1982

THIS DOCUMENT CONTAINS
CONFIDENTIAL INFORMATION

FEDERAL TRADE COMMISSION

STAFF REPORT ON THE CIGARETTE ADVERTISING INVESTIGATION

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May, 1981

> explicit and varied. Their constant repetition in advertising which reaches vast numbers of Americans of all ages must be viewed as significantly contributing to the portrayal of the desirability of smoking.[30]

Thus, the dominant themes of cigarette advertising are that smoking is associated with youthful vigor, good health, good looks and personal, social and professional acceptance and success, and that it is compatible with a wide range of athletic and healthful activities. One theme is conspicuously absent from all cigarette ads. Although these ads contain the required general warning, they make no mention of the numerous and specific adverse health consequences of using the advertised product.[31]

[illegible]

IV. ADVERTISING THEMES AS DESCRIBED BY THE MANUFACTURERS' MARKETING PLANS

The cigarette companies' documents reinforce the findings of the staff about the themes of cigarette advertising. For example, R.J. Reynolds' 1977 marketing plan for Salem states explicitly that:

> Motivational research has identified the phenomenon of image projection as a highly motivating force. Therefore, through the association of SALEM, and its brand styles with emulatable personalities and situational elements that are compatible with the aspirations and lifestyles of contemporary young adults, this important target segment will be attracted to the brand. Importantly, older smokers also relate

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[30] 1964 Cigarette Rule, supra, 20 Fed. Reg. at 8342.

[31] To the best of our knowledge, cigarette companies never have provided health information, other than the required warning and "tar" and nicotine figures, in their advertisements.

> favorably to this personality type; therefore, reinforcement of the current franchise is simultaneously achieved.[32]

The same marketing plan indicates that a primary theme for the promotion of Salem has been to associate the cigarette with the lifestyle of "young adult males" who are "masculine, contemporary, confident, self-assured, daring/adventurous, mature."[33] Marketing plans for other cigarettes are similar. A Doral campaign sought to project the image of "an independent, self-reliant, self-confident, take-charge kind of person."[34] A Winston man was projected as "a man's man who is strong, vigorous, confident, experienced, mature."[35]

Liggett & Myers' documents show that their Lark ads picturing a balloon high above land sought to give the consumer an association with "lightness and exhilaration."[36] L&M's campaign planned to position Lark as a "youthful, contemporary brand that satisfies the lifestyles of the modern smoking public." Its ads emphasize "moments of post-tension and relaxation."[37] The Eve smoker has been portrayed as a "sophisticated, up-to-date, youthful and active woman who seems to have distinct ideas about what she wants."[38]

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[32] Document A900022 - "Salem 1977 Annual Marketing Plan."

[33] Id.

[34] Document A900003 - "RJR Statement of Business 1977 - Doral Cigarettes."

[35] Document A900001 - "Winston King 1978 Marketing Plan."

[36] Document A900230 - "1974 Lark Annual Marketing Plan."

V. EFFORTS OF SOME CIGARETTE ADVERTISEMENTS TO DIVERT ATTENTION AWAY FROM THE HEALTH HAZARDS OF SMOKING

Many cigarette advertising techniques appear to denigrate or undercut the health warning. Information obtained from subpoenaed documents indicates that, at least in the case of several advertising campaigns, these techniques have been carefully planned. For example, documents from Brown & Williamson (B&W) and one of its advertising agencies, Ted Bates and Company, Inc., set forth the development of an advertising strategy for Viceroy cigarettes designed to suppress or minimize public concern about the health effects of smoking.

The documents show that, at the request of Ted Bates, a marketing and research firm conducted a number of focus group interviews on the subject of smoking in order to assist the ad agency in developing a marketable image for Viceroy cigarettes.[39] The final report summarizing the results of this research asserts that many smokers perceive the smoking habit as a "dirty" and dangerous one engaged in only by "very stupid people."[40] The report concludes:

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[37] Document A900251 - "1973 L&M Marketing Plan."

[38] Document A900245 - "1974 Eve Portfolio Test."

[39] Document A011345 - "An Action-Oriented Research Program For Discovering And Creating The Best Possible Image For Viceroy Cigarettes," prepared for Ted Bates Advertising in March 1975 by M. Kennan, Marketing and Research Counselors, Inc.

> Thus, the smokers have to face the fact that they are illogical, irrational and stupid. People find it hard to go throughout life with such negative presentation and evaluation of self. The saviours are the *rationalization* and the *repression* that end up and result in a defense mechanism that, as many of the defense mechanisms we use, has its own 'logic', its own rationale.
>
> * * * * * * * *
>
> Thus, smokers don't like to be reminded of the fact that they are illogical and irrational. They don't want to be reminded by either *direct* or *indirect* manner.[41]

The report proceeds to describe the elements of a good cigarette advertising campaign, in light of its findings, in a chapter entitled, "How To Reduce Objections To A Cigarette." The basic premise of the report's recommendations is that since there "are not any real, absolute, positive qualities and attributes in a cigarette," the most effective advertising is designed to *"reduce objections"* [42] to the product by presenting a picture or situation ambiguous enough to provide smokers with a rationale for their behavior and a means of repressing their health concerns about smoking. To provide a rationale for smoking, the ad must project the image that cigarettes provide the smoker with social acceptance, an acceptable means of rewarding himself or herself, a stimulant, a tranquilizer, a better self-image, etc. With regard to health issues, the report recommends: "Start out from the basic assumption that cigarette smoking is dangerous to your health - try to go around it in an elegant manner but don't try to fight it - it's a losing war."[43]

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[40] Document A901268 - May 26, 1975 "What Have We Learned From People? A Conceptual Summarization of 18 Focus Group Interviews On The Subject Of Smoking."

[41] *Id*. at 2, 3 (emphasis in original).

[42] *Id*. at 12 (emphasis in original).

One chapter of the report describes how the company can introduce "starters" to the Viceroy brand, a discussion which focuses almost exclusively on how to persuade young people to smoke. The report asserts:

> For the young smoker, the cigarette is not yet an integral part of life, of day-to-day life, in spite of the fact that they try to project the image of a regular, run-of-the-mill smoker. For them, a cigarette, and the whole smoking process, is part of the illicit pleasure category...In the young smoker's mind a cigarette falls into the same category with wine, beer, shaving, wearing a bra (or _purposely_ not wearing one), declaration of _independence_ and striving for self-identity. For the young starter, a cigarette is associated with introduction to sex life, with courtship, with smoking 'pot' and keeping late studying hours.[44]

The chapter then recommends a strategy for attracting young "starters" to cigarette smoking:

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[43] _Id_. at 17.

[44] _Id_. at 29-30 (emphasis in original).

> Thus, an attempt to reach young smokers, starters, should be based, among others, on the following major parameters:
>
> - Present the cigarette as one of a few initiations into the adult world.
> - Present the cigarette as part of the illicit pleasure category of products and activities.
>
> * * * * * * * *
>
> - In your ads create a situation taken from the day-to-day life of the young smoker but in an elegant manner have this situation touch on the basic symbols of the growing-up, maturity process.
> - To the best of your ability, (considering some legal constraints), relate the cigarette to 'pot', wine, beer, sex, etc.
> - Don't communicate health or health-related points.[45]

B&W adopted many of the ideas contained in this report in the development of a Viceroy advertising campaign. Thus, in a document entitled, "Viceroy Strategy," B&W notes repeatedly that its advertising campaign must provide consumers with a rationalization for smoking and a "means of repressing their health concerns about smoking a full flavor Viceroy."[46] The following excerpts from "Viceroy Strategy" are representative and indicate that in B&W's view, the other cigarette companies also have developed advertising strategies designed to cause repression of consumer health concerns about smoking:

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[45] *Id.* at 31.

[46] Document A015538 - "Viceroy Strategy," March 3, 1976, V.C. Broach, Group Project Manager, B&W (emphasis in original).

> Full flavor smokers perceive cigarette smoking as dangerous to their health...Given their awareness of the smoking and health situation, they are faced with the fact that they are behaving illogically. They respond to this inconsistency by providing themselves with either a rationalization for smoking, or, by repressing their perceptions of the possible dangers involved. To date, major full flavor brands have either consciously or unconsciously 'coped' with the smoking and health issues in advertising by appealing to repression. [emphasis added.]
>
> * * * * * * * * *
>
> The marketing efforts must cope with consumers' attitudes about smoking and health, either providing them a rationale for smoking a full flavor VICEROY or providing a means of repressing their concerns about smoking a full flavor VICEROY. [emphasis in original.]
>
> * * * * * * * * *
>
> Advertising Objective - To communicate effectively that VICEROY is a satisfying flavorful cigarette which young adult smokers enjoy, by providing them a rationalization for smoking, or, a repression of the health concern they appear to need.

B&W then describes its plan to accomplish its advertising objective. Three advertising strategies would be used:

> 1. The 'satisfaction' campaign provides a rationalization: VICEROY is so satisfying that smokers can smoke fewer cigarettes and still receive the satisfaction they want....
> 2. The 'tension release' campaign provides a rationalization: VICEROY'S satisfying flavor can help the smoker in a tense situation....
> 3. The 'feels good' campaign appeals to the smoker by repressing the concerns he may have about smoking by justification: If it feels good, do it; if it

feels good, smoke it....[47]

B&W documents also show that it translated the advice on how to attract young "starters" into an advertising campaign featuring young adults in situations that the vast majority of young people probably would experience and in situations demonstrating adherence to a "free and easy, hedonistic lifestyle."[48]

Other documents submitted by B&W show that the company has attempted to capitalize upon the erroneous consumer perception that there is a health benefit to smoking mentholated cigarettes. Documents pertaining to the marketing of Kool cigarettes demonstrate that the company is aware of the consumer misperception about the relative safety of menthol cigarettes and utilizes it in the development of advertising strategies for Kools.[49]

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[47] These strategies were employed in a six-month media campaign conducted in three test cities in 1976. The advertising allotment for the campaign was approximately ten times the normal advertising dollar amount for a six month period. (Document A015486 - Memorandum from M.M. Matteson to V.C. Broach, July 14, 1976, emphases added).

[48] Document A080115 - "Viceroy Marketing/Advertising Strategy," January 26, 1976.

[49] In 1976, B&W held four focus group discussions to gauge menthol smokers' responses to a new Kool 120mm cigarette. The majority of the participants were menthol cigarette smokers. In a number of cases, the participants told B&W that they switched to menthol either for health considerations or from a general feeling that menthol cigarettes are less dangerous. According to B&W, a "pseudo-health image" has accrued to mentholated cigarettes. (Document A080675 - "Low Tar Longs Project - Creative Agency Assignment," 1977.) By characterizing the health image of mentholated cigarettes "pseudo," B&W admits its knowledge that menthol is of no health benefit to (Continued)

A third set of documents obtained from Brown and Williamson reveal that in 1976, Brown and Williamson introduced a new brand of cigarette named Fact. The Brown and Williamson documents indicate that the company believed that Fact cigarettes were a new product which reduced the amount of harmful gas in the cigarette smoke inhaled by the consumer. Therefore, Fact was initially advertised as a brand with the unique ability to filter certain gases.[50] However, initial sales of Fact were not considered satisfactory by Brown and Williamson, so in 1977 it temporarily halted all advertising and promotion of the cigarette while it developed a new market strategy.[51]

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[49] (Footnote Continued)

smokers. In a 1978 document discussing Kool cigarettes' strengths and weaknesses, B&W also admits that one of Kool's strengths "rides on the connotation that menthol has health overtones." (Document A006981 - Memorandum from R.L. Johnson to F.E. McGowan, B&W, March 13, 1978.) In addition, B&W states that one of the strengths of its Kool Super Longs is that "menthol and 'tar' delivery has synergistic therapeutic implications." (Id.) B&W intends to exploit this false belief. In its document describing Kool's objectives through 1981, B&W states that its strategy will be to provide product safety reassurances while enhancing the satisfaction and refreshment perception. (Document A035669 - "Kool Three-Year Objectives," August 15, 1978.)

In fact, mentholated cigarettes tend to have a high "tar" and nicotine content.

[50] Document 7244 - "Fact 1976 Concept Description and Potential and Marketing Plan."

[51] Document 35523 - "Fact 1977 Repositioning and 1978 Marketing Plan Summary."

In April 1977, Brown and Williamson's advertising agency, Post-Keyes-Gardner, Inc., presented Brown and Williamson with its marketing and strategy recommendations for the reintroduction of Fact cigarettes.[52] The ad agency proposed two possible strategies to distinguish Fact from other cigarettes: 1) "More complete health protection through selective gas filtration," 2) "More taste and satisfaction in a low tar cigarette."[53] About the proposed strategy focusing on better health protection as the result of inhaling lower levels of gas, Post-Keyes-Gardner, Inc. wrote:

> A secondary opportunity to distinguish Fact from the mass of tar number claims is by capitalizing on the product's unique selective gas filtration. This would demand "product image" advertising and would provide the brand with a real point of difference. It would mean expanding the cigarette health issue beyond tar to encompass gas. However, this would require establishing "gas" as a meaningful health hazard in cigarettes because currently there is very low consumer awareness or comprehension of the gas problem. The Agency believes one of the major problems with the introductory advertising for Fact was that it failed to educate health concerned consumers about the dangers of gas. This failure to establish the gas problem meant that Fact's selective filtration promise was meaningless to the majority of the target audience. However, if smokers are effectively educated regarding this problem, the selective gas filtration promise may still be powerful, particularly among the very health conscious.[54]

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[52] Document 35524 - Brown & Williamson, "Marketing Advertising Strategy Recommendations for the Reintroduction of Fact Cigarettes," April 18, 1977. (The same document was also submitted by Post-Keyes & Gardner, Inc. - Document 714569).

[53] Id.

[54] Id., at 3.

However, the agency also noted the weakness of this proposed strategy:

> This strategic option assumes gas will become a major health issue. To ensure it becomes an issue will require an educational approach in introductory advertising. It is questionable whether any cigarette manufacturer should be publicizing a new health hazard for cigarette smokers. The desire to avoid spelling out the gas hazard in advertising could severely weaken the effectiveness of this approach.[55]

Ultimately, Brown and Williamson documents indicate that it elected not to educate the public about the health hazards associated with the gases in cigarette smoke and not to focus the Fact ad campaign on the low gas issue. The reason for Brown and Williamson's decision is explained in a document entitled "Fact 1977 Repositioning and 1978 Marketing Summary":

> Until the problem of gas becomes public knowledge through government investigation or media coverage, a low gas benefit will remain of little strategic value. [emphasis added][56]

The rationale is restated in a memorandum from the representative of the Brown and Williamson "Brand Group" which had overall responsibility for Fact:

> We do not support definition in advertising of the problem of gas in order to specifically communicate its consumer benefit and distinguish it from low "tar". To supply such definition would require overt references to the alleged ciliatoxic and cardiovascular ill effects of smoking. The possible ramifications of this in the Legal, Regulatory, and Policy areas are appelling...a likely result of such

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[55] *Id.*, at 6.

[56] Document 35523 - "Fact 1977 Repositioning and 1978 Marketing Plan Summary."

March 24, 1982

STATEMENT
of the
BAKERY, CONFECTIONERY & TOBACCO WORKERS INTERNATIONAL UNION
to the
SENATE COMMITTEE ON LABOR AND HUMAN RESOURCES

Re: S. 1929, "The Comprehensive Smoking Prevention Education Act of 1982"

The Bakery, Confectionery and Tobacco Workers International Union represents over 160,000 workers in the United States and Canada. Approximately 30,000 of our members are employed in the U.S. tobacco industry.

We present this testimony on behalf of all of our members - those who are employed in that industry, as well as those employed in the various aspects of the food production industry. We also speak on behalf of the many workers in the tobacco industry who are not organized and do not, therefore, have a collective voice to represent them at the hearings.

We oppose S. 1929, The Comprehensive Smoking Prevention Education Act, for two very important reasons.

First, it threatens our industry and our workers with the needless loss of sales, earnings, and ultimately, jobs.

Second, it threatens a host of other workers in other industries and the general public with the loss of protection against hazardous environmental and occupational exposures.

To put it plainly, this legislation is not what it seems to be. It is not merely a harmless labeling bill, but rather the first step down the road to prohibition. In fact, it is not merely a tobacco and health bill, but rather a red herring that could be used to divert attention from efforts to undermine other health policies and programs.

Mr. Chairman, this bill masquerades as labeling legislation, but there is an issue behind the issue. The hidden issue is whether national policy should shift from education to prevention, from choice to coercion.

This Committee will have to decide between those two roles for the government. Should the government continue to give people information so they can make their own free choice? Or should it aggressively persuade individuals to modify their behavior until they stop?

Present U.S. policy calls for the public to be informed, the measure of effectiveness being the extent of public awareness. Awareness stands at an astonishingly high level of 90 percent, verging on universal acceptance according to behavioral scientists.

The neo-prohibitionist strategy of this bill calls for prevention, and is based on the theory that if people reject the government's admonitions, they cannot really be informed -- and, therefore, must be reformed. The new measure of effectiveness shifts from knowledge to conformity.

Even on these terms, the present policy of education is working! The prevalence of smoking has dropped to 35 percent, the lowest ever recorded by the Gallup Poll. But apparently, it is not enough that awareness is at its highest level, and smoking, the disapproved behavior, is at its lowest point.

That more prevention is nevertheless prescribed betrays the neo-prohibitionist motivation that lies just under the surface.

This motivation explains why the bill loads packages and advertising with more warnings and lists than any other product is required to carry. It also explains why the bill opens the door to a massive overload of litigation. If these prohibitionist effects succeed in depressing sales by just one percent, the adverse impact would be significant. Based on data from a recent Wharton study of the tobacco industry's contribution to the U.S. economy, we estimate that the loss for just nine of the states (California, Illinois, Kentucky, New Jersey, New York, North Carolina, Ohio, Pennsylvania, Texas) would be more than 10,000 jobs and more than $170 million in wages. One-fifth of this loss would come from tobacco farming, manufacturing, retail sales, and suppliers; the remainder would result from the ripple effect on the rest of the economy.

Further economic hardship would result from the loss of export markets and of American jobs that depend on exports. This bill goes far beyond existing legislation by requiring warning labels on cigarettes produced in this country for export. Could American cigarettes carrying a health warning label compete with cigarettes which bear none? Can there be any doubt of the result on sales and on American jobs?

The American Cancer Society, a major lobbying force for this legislation, wants to cause more than a one percent drop in smoking. Several years ago, they launched their Target Five campaign, aimed at a 25% decrease in smoking in five years. And, it must be recognized that in Sweden, the source of this bill, the government's stated goal is to achieve a smoke-free nation by the year 2000.

We seriously question the wisdom of buying the Swedish import for Americans. We seriously question the wisdom of disrupting a health industry and creating more unemployment in a recession.

We question the wisdom of setting up a new anti-smoking bureaucracy with unspecified spending authority when other essential health and social programs are being slashed. Mr. Chairman, we do more than "seriously question the wisdom" of this bill -- we reject its folly.

Now, let us turn to the second major ground for our opposition.

Section 3 deals with so-called findings. These blame every major chronic disease on smoking, and thereby create a smoke-screen for the occupational and environmental factors involved.

The very first one states that "the Congress finds that cigarette smoking is the largest preventable cause of illness and premature death in the United States and is associated with the unnecessary deaths of over three hundred thousand Americans annually."

At first glance, it is difficult to conceive of a statement more alarming, more compelling, more demanding of remedial action. It calls for nothing short of outlawing tobacco.

But on reflection, this finding is curiously phrased; the words have an Alice-in-Wonderland quality. Their meaning is hard to pin down. For example, if cigarette smoking is the "largest preventable cause of illness," what are the second and third largest preventable causes? What are the "non-preventable" causes? Is smoking "preventable" while environmental pollution is not?

If these statements of findings had to be substantiated, as the FTC requires advertising statements to be, I doubt they would survive. The bill says flatly that smoking "is associated with" over 300,000 deaths a year. Yet, the first Surgeon General's Report in 1964 stated that:

> The total number of excess deaths causally related to cigarette smoking in the U.S. population cannot be accurately estimated.

The Committee which wrote the report considered the possibility of trying to make such calculations, but rejected the idea because "it involves making so many assumptions that the Committee felt that it should not attempt this..."

That restraint is as needed now as it was then.

We believe the findings in this bill are unsubstantiated and will be misused to the detriment of millions of workers exposed to occupational hazards. We oppose this bill and its findings to show our solidarity with:

- Coal miners whose black lung disease has been blamed on smoking.
- Textile workers whose brown lung disease has been blamed on smoking.
- Asbestos workers whose lung diseases have been blamed on smoking.

And the list includes uranium workers, chemical workers, metal workers, shipyard workers, and many others. Public Health Service and the voluntary health organizations should honestly abandon the blame-the-victim approach and get at the truths of what is causing disease.

Earlier this year, the Congress heard testimony from two scientists who reported that "at least 11 percent and more likely 21 percent" of lung cancer in the U.S. can be attributed to air pollution. They noted that the proportion of adult smokers has decreased and that cigarettes now contain half the tar content of 20 years ago, yet lung cancer rates continue to climb. "To us this indicates that something else is at work," the scientists said.

But to the supporters of this bill, it's all cigarette smoking.

Recently, the National Wildlife Federation reported that "responsible scientists believe air pollution is responsible for about 50,000 excess deaths, seven million sick days, and 15 million days of restricted activity per year."

But to supporters of this bill, it's all cigarette smoking.

The lung cancer rate for white men along the coastal sections of Northern Florida, South Carolina and Georgia are among the highest in the nation. The National Cancer Institute says it may be the result of exposure in the booming shipbuilding industry during World War II. A Florida State University study attributes it to airborne chemical pollutants from industrial plants in New York and Illinois.

But to the supporters of this bill, it's all cigarette smoking.

The New York Times has recently looked at the growing controversy over whether environmental or lifestyle factors cause cancer. The former chief epidemiologist of the American Cancer Society, and others associated with the industrial establishment, believe that cancer-causing pollutants are relatively minor factors compared to factors such as smoking, diet, alcohol, and even sexual and reproductive behavior, a view that is consistent with the bill's "findings".

But the New York Times also reported that other scientists believe that factors other than smoking are involved; they are worried about "poisons escaping from smokestacks, toxic waste dumps, nuclear reactors." They are worried that "black men smoke less than whites, yet have higher lung cancer rates, perhaps because they have...more hazardous jobs."

But the supporters of the bill have no similar worries; to them, it's all due to smoking.

Politics makes strange bedfellows. The House version of this Act has been introduced by a member with a 100% AFL-CIO voting record;

S. 1929 has been introduced by a man with a zero AFL-CIO rating. We can't split the difference. We totally oppose both bills.

And in doing so, I would like to point to a report adopted by the AFL-CIO Executive Council in 1980:

> Some employers have exploited scientific studies of the combined effects of smoking with occupational exposure to toxic substances and conclude that it would be unnecessary to control exposure of these substances, if workers stopped smoking.
>
> The AFL-CIO is opposed to any coercive efforts to infringe on individual rights of individuals who smoke or of those who don't. We also oppose misuse of scientific data concerning smoking and exposure to toxic substances to serve as a rationale for failure to take necessary steps to prevent worker exposure to toxic substances in the workplace, which are shown to adversely affect their health.

We are impressed by the foresight of the Executive Council in stating two years ago the fundamental basis for rejecting this legislation today.

Comments on H. R. 4957 - - Proposed
S. 1929
"Comprehensive Smoking Prevention Education Act of 1981"

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The proposed bill, H.R. 4957, states that "Congress finds that ... smoking is the major cause of chronic obstructive lung diseases such as emphysema" [Section 2 (2)]. The statement paraphrases one sentence in the introduction of the Chapter on non-neoplastic bronchopulmonary diseases of the 1979 Report of the Surgeon General on Smoking and Health, which reads "cigarette smoking is the most important cause" (1). However, a careful examination of the chapter raises questions about the validity of that statement because of the complexity of the causation of chronic obstructive lung diseases in general and of pulmonary emphysema in particular. Although there are a multiplicity of suspected pathogenic mechanisms and of etiologic factors, the Surgeon General's Report refers only to those relating to cigarette smoking with an incidental discussion of outdoor pollution, indoor pollution, infections and familial or genetic predisposition. The author of this submission intends to discuss his personal research investigations and those of others questioning the conclusion that smoking is either "the major cause" or "the most important cause" of chronic obstructive lung diseases. Briefly, his reasons are as follows:

(a) Animal experiments have failed to reproduce pulmonary emphysema from long-term exposure to cigarette smoke, although this has been successfully done so for sulfur oxides, nitrogen oxides and other inhalants.

(b) Functional and histopathological studies in humans show uncertain and inconsistent effects of cigarette smoking that do not support the causal relationship between cigarette smoking and chronic obstructive lung disease.

(c) Epidemiologic studies suggest the association of chronic obstructive disease with several risk factors, such as levels of outdoor and indoor pollution, alcohol consumption, occurrence of previous infections, familial predisposition and genetic susceptibility.

A. Experimental Animal Studies on Pulmonary Emphysema.

From 1948 to 1977, the author and his colleagues conducted smoking-related research at the University of Pennsylvania School of Medicine where he was a Professor of Pharmacology. The nature of this investigation was guided in part by comments in the 1964 Surgeon General's Report on Smoking and Health which suggested the need for definitive data on the relationship between cigarette smoking and pulmonary emphysema (2). Although eight of the author's publications were mentioned in the 1967, 1968, 1969 and 1971 Surgeon General's Reports, the coverage was brief and out of context (3-6). Over thirty of his articles on cigarette smoking and over a hundred articles published in the 1970's on related subjects were not mentioned.The government writers cannot be criticized entirely, however, because they have been examining only "Smoking and Health" rather than conducting a global review of all suspected risk factors of chronic obstructive lung diseases. Yet this isolated emphasis on one factor has limited the usefulness of Surgeon General Reports as scientific documents.

a. Experimental Pulmonary Emphysema in the Dog. The 1981 report of the Surgeon General (7) states that "there are no published studies that acceptably show in an animal model that the development of emphysema is induced by cigarette smoking. ... One study in which dogs received smoke directly through chronic tracheotomies reported the development of emphysema [Auerbach, Hammond, Kirman, Garfinkel and Stout (1967)]. The lesions were not conclusive and the results have not been confirmed by others."

The initial series of studies conducted by the author and his colleagues dealt with the effects of cigarette smoke in dogs (8-17). These studies failed to show that inhalation of cigarette smoke caused pulmonary emphysema. It is therefore not surprising that there has been no confirmation of a single study reporting the development of experimental emphysema. It should be noted that the single positive study, although questioned in the 1981 Surgeon General's Report, was highlighted in earlier Reports (including 1979) and may have been responsible for the impression that cigarette smoking is an established cause of pulmonary emphysema.

b. Experimental Pulmonary Emphysema in the Rat. In 1967, the author and his collaborators developed an experimental model for producing pulmonary emphysema in rats (18). Unlike other models reported hitherto, this model permitted functional measurements in addition to histopathologic observations signifying pulmonary emphysema. The exposure of animals to cigarette smoke did not cause pulmonary emphysema (19). Other investigators have also used the same model and have confirmed essentially its acceptability for the study of pulmonary emphysema.

c. Experimental Pulmonary Emphysema in the Mouse. In 1974, the author and his collaborators developed a technique for measuring lung function in the mouse that could not be performed previously (20-22). Chronic exposure to cigarette smoke simulating the dosage of smokers did not cause pulmonary changes signifying emphysema. It was a disappointment to the author that the only study comparing high and low nicotine cigarettes on pulmonary function of mice was overlooked in the 1981 Report of the Surgeon General entitled The Changing Cigarette (7). It is possible that the study was ignored because of the unexpected observations that the lungs seem to adapt to repeated exposure of cigarette smoke.

d. Comparative Studies with Air Pollutants. Dogs, rats and mice are useful experimental models for the study of toxicity of air pollutants. Animals exposed to low levels of products of fuel combustion show impaired lung function and pathological lesions characteristic of pulmonary emphysema and bronchitis. The studies are described in the new series of Criteria Documents that have been prepared by the Environmental Protection Agency on ozone and photochemical oxidants (23), nitrogen oxides (24) and sulfur oxides with particulates (25). This author questions the logic of finding cigarette smoke "the major cause" of pulmonary emphysema when primary air pollutants have been shown to cause pulmonary emphysema in experimental animals, and with the same models, cigarette smoking has not.

B. Human Studies on Pulmonary Function and Histopathologic Changes in Cigarette Smokers.

The Chapter on chronic obstructive lung diseases in the 1979 Surgeon General's Report (pages 6-7 to 6-52) starts by defining the terms chronic bronchitis and pulmonary emphysema. For each definition, there is a positive statement on structural or pathological alterations such as hypertrophy of mucous secreting apparatus and epithelial metaplasia as well as more classic histopathological evidence of inflammation for bronchitis; and abnormal enlargement of the air spaces distal to the terminal nonrespiratory bronchiole accompanied by destructive changes of the alveolar walls for emphysema.

These definitions are completely ignored in the remainder of the chapter because functional tests, macrophage changes and enzymatic contents are regarded as pathological or adverse signs of chronic bronchitis or pulmonary emphysema. The definition of adverse and nonadverse effects has been vigorously debated in recent years by the National Academy of Sciences (26) and the World Health Organization (27, 28). The National Academy of Sciences defines adverse effects as responses that are irreversible; the reversible effects are regarded as adaptation or defense mechanism of the lung in response to the inhalant. The predominant opinion is to establish irreversibility as a prerequisite to the definition of an adverse effect. In terms of tobacco use, most of the functional effects described in the 1979 Surgeon General's Report are reversible, not adverse in nature, and are indicative of physiological and biochemical adaptation of the lungs.

a. Respiratory symptoms and pulmonary functional changes. The section on the natural history of chronic obstructive lung disease (pages 6-10 to 6-11) ends with the qualifying statement that pathological data are the most specific and sensitive parameters relating to pulmonary emphysema or chronic bronchitis. It is further stated that "the relationship of early respiratory symptoms to subsequent development of lung disease is unclear" and that "longitudinal studies demonstrating that individuals with abnormal tests of small airways function are at greater risk for lung disease are unavailable". Respiratory symptoms and small airway functional tests therefore are not necessarily indicators of adverse effects or pathological processes.

That smokers show abnormal lung function has not been uniformly observed. In addition to a single study mentioned in the Surgeon General's Report, there are five other publications during the late 1970's that were not cited (29-33).

b. Clearance Mechanisms. The discussion of mucociliary transport (page 6-32) is another example of selective citations from the literature showing negative aspects of cigarette smoking, and omission of conflicting results. Articles that show smoking accelerates bronchial clearance in humans have been omitted. The studies of Albert et al on accelerated clearance in donkeys have been cited, but their article on human clearance stimulation in smokers (34) has been omitted, thus giving readers the impression that the phenomenon has only been seen in donkeys.

Camner, who pioneered in clearance studies in man and whose technique has been adapted by many clinical pulmonary physiologists, has been overlooked. The basic concept that has evolved from Camner's work is that adrenergic stimulation increases mucociliary transport (35), an effect to be expected from cigarette smoking. Patients with predominant pulmonary emphysema but without chronic bronchitis are reported to have no impairment of mucociliary transport (36). Therefore, it is unlikely that smoking, by accelerating airway clearance, would contribute to the pathogenesis of emphysema.

c. Proteolytic lung damage. The hypothesis that cigarette smoke causes a protease-antiprotease imbalance and in turn potentially leads to pulmonary emphysema is based on a group of selected observations. There is no discussion as to why the hypothesis has not been generally accepted. Turino (37), the first researcher to show that proteolytic enzymes influence the pulmonary mechanics in vivo, has noted flaws in the hypothesis that emphysema is caused by the imbalance of protease and antiprotease:

> "An inconsistency for the primary role of elastin alteration as a mechanism for inducing pulmonary emphysema has been a failure to demonstrate alterations in elastin content or composition in human emphysema. Morphologically, elastin fibers have appeared disrupted and diminished, yet most data from human lungs (Johnson and Andres 1970; Pierce and Hocott 1960; and Pierce et al 1961) with the exception of that of Briscoe and Loring (1958), have shown no reduction of parenchymal elastin content by gravimetric techniques."

The above comments can be reinforced by the 1976 observations of Kuhn et al (38) that hamsters treated with elastase intratracheally and developing emphysema also show normal elastin content. Even lung fluid samples collected from smokers and nonsmokers showed no difference in proteolytic enzyme content (38a).

C. Epidemiologic Studies.

Increased mortality from pulmonary emphysema and/or chronic bronchitis among smokers compared to nonsmokers is suggested by seven prospective studies summarized in the 1979 Report (page 6-10). However, these studies are far from being consistent and tend to suggest that many risk factors including cigarette smoking need to be considered in studying the etiology of chronic obstructive lung disease.

a. Air Pollution. The role of air pollution as a risk factor in chronic obstructive lung disease has been the subject of several publications not cited in the 1979 Report. Studies in Tucson, Arizona (39) and in Busselton, Australia (40) showed that an urban environment may contribute to the normal increase in closing volume with age and to the incidence of respiratory symptoms. The higher levels of pollution in England than in the United States has been cited as a cause of the lower ventilatory functional measurements among British males (41).

b. Socioeconomic Status. The illustrative table presented in the 1979 Report (page 6-39) is based on a survey by Higgins of residents of Tecumseh, Michigan. The overall prevalence of chronic bronchitis for various occupational groups ranged from 12.3 for professionals and managers to 30.0 for laborers. An uncited 1977 survey by Lebowitz (42) is more accurate because the diagnosis of emphysema and chronic bronchitis was confirmed by medical examination. His most important conclusion is that smokers do not have a higher incidence of chronic obstructive lung diseases than nonsmokers or exsmokers when the adults were grouped according to income and educational backgrounds. Education and income were inversely related to prevalence of obstructive lung diseases even after controlling for sex, age and smoking.

c. Alpha$_1$antitrypsin deficiency. The summary statement in the 1979 Report that individuals with severe deficiency have the onset of symptomatic chronic obstructive lung disease probably "abbreviated by smoking" was based on selected citations. Two publications in Chapter 6 were not accurately quoted. Chan-Yueng et al and Morse et al did not only examine subjects with mild deficiency but also individuals with severe antitrypsin deficiency which did not deteriorate with smoking. Kidokoro et al (43) concluded in their study of subjects with severe deficiency that "variables other than age and cigarette smoking, influence the severity of emphysema."

d. Other genetic and familial factors. The limited discussion in the 1979 Report needs expansion due to increasing evidence of familial aggregation of chronic obstructive lung disease. The studies of familial prevalence of lung disease conducted by Cohen et al clearly indicate that there is a familial factor unrelated to cigarette smoking and antitrypsin deficiency that is involved in chronic obstructive pulmonary disease (44).

Familial aggregation of chronic bronchitis independent of cigarette smoking, sex, respiratory illness history, residence in common household, geographical distribution and antitrypsin variants has also been reported by Tager et al (45) in 1978. There has also been revived interest in the concept of a congenital or developmental etiology of pulmonary emphysema (46). Additional research directed at the basic cause of emphysema may lead to the identification of additional genetic factors that result in developmental abnormalities leading to adult pulmonary emphysema.

e. Infections. A recent report on the Tecumseh study reveals that acute infection may play an independent role in the pathogenesis of chronic respiratory disease (47). For serologic infection rates for three viruses and M. pneumoniae in males, the incidence of bronchitis is higher for nonsmokers (23%) than in smokers (8.8%). Lebowitz and Burrows (48) arrived at a similar conclusion, i.e. acute respiratory illness is a major risk factor in the etiology of chronic obstructive lung disease.

f. Alcohol consumption. Since 1977, there have been several reports that consumption of alcohol is associated with higher incidence of chronic bronchitis (49-51). In the Tucson study, Lebowitz (52) has concluded that alcoholism is an independent risk factor for chronic obstructive lung disease. There are also earlier observations that alcohol feeding in rats causes pulmonary cellular changes, depression of immunologic reaction, and increased susceptibility to infection (53-55).

D. CONCLUDING REMARKS

It is the opinion of the author of this submission that cigarette smoking is not "the major cause" of chronic obstructive lung diseases, such as pulmonary emphysema. This opinion is based on the author's own research studies and his interpretation of literature on the subject. It is clear that additional research is needed to determine which of the different risk factors for chronic obstructive lung disease are important in a causal sense.

Domingo M. Aviado, M.D.

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March 8, 1982

Cigarette Smoking of Pregnant Women

A note regarding the "Comprehensive Smoking Prevention Education Act of 1982"

HR 5653
S. 1929

My name is Bea J. van den Berg. Currently I am director of the Child Health and Development Studies, a research unit of the School of Public Health of the University of California at Berkeley.

I received my medical doctor's degree in the Netherlands and in 1965 I joined the research staff of the Child Health and Development Studies that were designed and directed by the later Dr. J. Yerushalmy, professor of Biostatistics at the University of California at Berkeley. My CV is enclosed with the copy of my statement.

I am here to express my concern about the statements made in the proposed "Smoking Prevention Education Act of 1982" regarding an increased risk of spontaneous abortion, stillbirth, premature birth, child weight deficiencies and birth defects in pregnant women who smoke.

For many years I have been involved in research regarding pregnancy and pregnancy outcome, and research results do not support these statements.

Our studies were among the very first to identify the lower birth weight of babies born to smoking mothers as compared with that of non-smoking mothers. However, our studies did not indicate an increased risk of abortion and still births among smoking pregnant women nor are our studies supportive of the stated increased risk of birth defects.

For almost two decades, at the Child Health and Development Studies, we have studied the pregnancy outcome of women who smoked cigarettes during pregnancy, in comparison with women who never smoked or who stopped smoking before or early in their pregnancy. These studies were based on interviews, early in their pregnancies, of some 15,000 women who were members of Kaiser Foundation Health Plan, and who enrolled in the Child Health and Development Studies. Extensive information was obtained from the medical records of the mothers during their pregnancy and delivery and from the medical records of the children from birth to at least age five.

Our studies are prospective and observational; prospective because the smoking data were assembled before the outcome of the pregnancy was known. This approach avoids a possible recall bias that might occur when the mothers are interviewed after the termination of the pregnancy. An unfavorable pregnancy outcome might affect the mothers' recall of any event that occurred during her pregnancy, including smoking habits. Our studies are observational and not experimental; this signifies that not the researcher, but the women themselves decide whether or not to smoke cigarettes. The women in these separate groups might be different, not only in their smoking habits, but also in various other known or unknown characteristics. This methodological problem, coined by Dr. J. Yerushalmy as the problem of self-selection, has to be taken into consideration if we compare the pregnancy outcome of women who do smoke with the pregnancy outcome of women who do not smoke.

I would like to summarize the results of our studies to date that relate to the statements concerning pregnancy in the proposed Bill.

On birth weight. As early as in 1962 Dr. Yerushalmy reported an increased proportion of low birth weight infants among the offspring of smoking pregnant women as compared with the offspring of non-smoking pregnant women. The observed association may indicate a cause-and-effect relationship but it may also be influenced by differences between smokers and non-smokers in characteristics other than smoking. A later study indeed showed that a variety of other maternal characteristics, such as socioeconomic status, maternal age and ethnic background, influence the association between smoking during pregnancy and birthweight of offspring. Controlling for a number of these variables reduces the crude difference in the birthweight of the offspring of both groups of women.

Yerushalmy's later work on maternal smoking was aimed at developing methodologies and designing studies to test the causal hypothesis against the alternative hypothesis that increased incidence of low birth weight is due to the differences between smoking and non-smoking pregnant women. One such study evaluated the frequency of low birth weight among the offspring of mothers who began smoking after the birth of the infants. It was found that women who subsequently became smokers also had a high incidence of low birth weight babies during the period before they started to smoke. This finding cannot be explained by the causal hypothesis and underscores the need for a larger study to confirm or refute this finding.

On abortion and stillbirth. Our statistical study, comparing for each week of gestation the risk of fetal death for smoking and non-smoking pregnant women, failed to find a difference between the two groups. These results, based on the pregnancy outcome of 15,000 pregnant women, do not provide evidence that smoking during pregnancy raises the risk of spontaneous abortion and stillbirth.

On birth defects. We recently published the results of our evaluation of the incidence of congenital anomalies (or birth defects) among the 14,735 children born to women who never smoked, to women who were past smokers or to women who smoked during pregnancy. The children were offspring of mothers enrolled in the Child Health and Development Studies. Our data did not show a difference in the incidence of severe congenital anomalies among children of smoking women and children of women who never smoked. We also looked at the incidents of non-severe congenital anomalies. We found that, while the incidence of these non-severe birth defects among children of heavy smokers (more than 20 cigarettes per day) was slightly more than among children of non-smokers, the incidence among children our light smokers (less than 20 cigarettes per day) was equal to that of the children of non-smokers. These observations, again, do not support the statement in the proposed Bill that cigarette smoking by pregnant women may result in birth defects.

In conclusion, our data do not support the statements in the proposed Bill.

March 5, 1982

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I am a sociologist, currently a University Professor at Boston University. The following is a personal statement, which does not necessarily represent the views of any institution with which I am affiliated. (A brief vita is enclosed.)

My attention was first drawn to H.R. 4957 and S. 1929 by an article in the New York Times on January 30, 1982; I subsequently obtained the texts of the two bills. The latter, in my opinion, constitute a further step in a development that has, for some time now, interested me as a sociologist and disturbed me as a citizen; that is, the growing militancy of the anti-smoking movement and its attempts to control or limit smoking activity.

I have no competence with regard to the medical questions at issue. However, I do claim competence with regard to the broader social and cultural context of this development, and it seems to me that it raises some rather fundamental questions about the scope and purposes of democratic governance on which any citizen has the right to claim competence.

What first interested me in this matter was the aggressiveness with which anti-smoking activities carried on their campaign. It annoyed me as a smoker (though perhaps I should say that I smoke cigars and pipes, not cigarettes, which are the major targets of the anti-smoking groups). No one likes to be depicted as a victim of debilitating vice or a walking threat to public health, and the segregation increasingly imposed on smokers in various public places is frequently demeaning. What also intrigued me, though, was _why_ some anti-smokers were so aggressive about this matter. I could not quite believe that they were all that concerned about _me_ (and other practitioners of this custom). After all, a lot of other things that I and others do are also claimed to be bad for health, yet no comparable furor surrounds them -- and I began to suspect that more complicated motives and interests were involved. I expressed these misgivings in the attached article, "Gilgamesh on the Washington Shuttle," published in _Worldview_ magazine in November 1977.

While this matter has not been a major focus of my professional activities (which, in recent years, have centered on the problems of Third World development), I have maintained an interest in the issue of smoking, contemporary culture and public policy. In the summer of 1979, for example, I attended the Fourth World Conference on Smoking and Health, held in Stockholm under the auspices of the World Health Organization (no doubt an appropriate location considering that the Scandinavian countries have the most stringent anti-smoking policies anywhere). I subsequently served as consultant to a study of the anti-smoking movement in Britain and the United States conducted by Professor Aaron Wildavsky of the University of California at Berkeley. In 1981 I gave a presentation at the Conference on Consumer Policy at the Wharton School in Pennsylvania. (This dealt generally with the evaluation of risks in everyday life; my presentation only mentioned smoking in passing, but it placed the smoking issue in a wider context of the quest for risk reduction.) Consequently, I have a good measure of familiarity with the various arguments made in the course of this controversy.

As to the bills under consideration here, I have no doubts whatever about the sincere intentions of the sponsors. What concerns me is the manner in which such legislation, if enacted, could be used by the anti-smoking forces. My concern is that this would be another step in a long-term campaign of stigmatizing and even criminalizing smoking. First, smoking is stigmatized as a disease; then, smokers are physically segregated; finally, smoking would be viewed as abnormal behavior, to be eventually eradicated or suppressed in all but the most private locations (to be engaged in, if at all, by consenting adults in the privacy of their bedrooms). But what concerns me even more is what this development says about the role of government (in this case, the Federal government) in our lives. It seems to me that those who favor or are considering this legislative proposal should give serious thought to two matters -- the general cultural context and the issue of government power.

The General Cultural Context

The campaign against smoking is not an isolated phenomenon; rather, it must be seen in a much wider context of cultural and social developments in this society. Specifically, it is part of a pervasive quest for security on the part of large numbers of Americans (who, incidentally, share this trait with people in other Western societies). There is a mind-set in this that cannot be unfairly described as institutionalized hypochondria: Life is full of hidden dangers and risks, many of them imposed on the innocent citizenry by rapacious forces beyond their control; this is intolerable; citizens must, therefore, organize to combat these evil forces and, wherever possible, enlist government in this battle. The obverse of this vision of an infinitely dangerous environment is a utopia in which dangers and risks are reduced to a minimum if not eliminated altogether.

The combination of these two themes, one hypochondriacal and the other utopian, makes for a curious ambivalence of timidity and aggression. On the one hand, people with this mind-set see themselves constantly surrounded by terrible perils, many of them deliberately caused or exploited by forces perceived as enemies; on the other hand, the same people become increasingly combative, aggressive and single-minded in the pursuit of their own goals.

One can find this constellation of attitudes in a considerable variety of public issues. But, logically enough, health is a favored focus of these concerns. It is also logical (at least in a psychological sense) that individuals greatly concerned about their own health should be attracted to the anti-smoking movement. Now, I am not saying that it is irrational to be concerned with health; of course it's not. But in the mind-set at issue, there is a tendency to deny that human life, by its very nature, is risky, and to assume that a lifestyle of passivity would eradicate all risks. As Wildavsky put it well, however, the greatest risk is to take no risks at all. Moreover, the ultimate human risk is death, and that, alas, cannot be avoided. Thus, the utopia of a risk-free existence finally denies the root fact of our mortality. All these attitudes are prominent in the anti-smoking movement. Reading the latter's literature, one gets the impression that, if we only stopped smoking, we would live forever. For example, there is the recurring phrase of "unnecessary deaths" (allegedly caused by the "smoking epidemic") -- a very curious and revealing phrase since, in the end, death is the final necessity for all of us.

I do not want to be misunderstood on this point. I'm not saying that everyone who favors public policies directed against smoking is motivated by such considerations. I am saying that these are themes in the ideology of the anti-smoking movement. I'm also saying that these themes are debilitating both to individuals and to society as a whole. Individuals and societies that dwell on the fearful dangers besetting them will tend to avoid risks of any kind, which is not a healthy posture in the face of the challenges of life.

There is another very important point to be made about the general context of this issue: There are significant class differences both in the general themes just described and specifically in the area of smoking. Lower-income people are more likely to smoke more than upper-income people; conversely, the anti-smoking movement (as are other movements concerned with health and risk reduction of all sorts) is overwhelmingly upper-income in its constituency. Putting this in more sociological terms, the anti-smoking campaign is an initiative coming from the upper-middle-class, while its putative beneficiaries are concentrated in the working class. Put in non-sociological terms, this is but another case of evangelism, in which the better-off classes in our society are trying to impose their own lifestyles on those who are less well off.

There is a long history of this, going all the way back to the early 19th century. The most ambitious case, of course, was Prohibition -- and the long Temperance Movement that preceded it. Here too, well-meaning middle-class people were trying to convert and eventually coerce their social "inferiors". In allusion to George Bernard Shaw's famous play, we might call this kind of cultural imperialism the "Major Barbara complex". Needless to say, working-class people resent this sort of meddling, and they resist it. The same tensions between well-meaning yet aggressive missionaries of a "nice" lifestyle on the one hand and those who are supposed to be the beneficiaries of these efforts on the other hand can be found in the contemporary smoking controversy. It also goes without saying that the upper-middle-class missionaries have greater resources at their disposal in this cultural welfare -- not only resources of money and power, but of education that supposedly legitimatizes their claims to "know better". The bottom line of this pretension is always "We know what is best for you and, therefore, we have the right to tell you how you should live." In everyday situations, the response of working-class people to such elitist interventionism tends to be quite forceful (and frequently unprintable).

Cultural themes are typically related to vested interests of one sort or another. The Temperance Movement kept thundering against the vested interests of the liquor merchants, but in time it was quite correct to speak of "Temperance interests". Similarly, the anti-smoking movement keeps attacking the "smoking interests" (which are, of course, identical with the tobacco interests); but by now one may also speak of "anti-smoking interests" -- that is, the vested interests of those who stand to gain (be it in terms of privilege or power) from the campaign against the regulation of, or the litigation involving smoking. Vested interests, inevitably, have different interpretations of available data. How the evidence may look to an absolutely disinterested observer (say, a non-smoking scientist from outer space) is an altogether different question, but one that, I think, has little to do with the political dynamics of the situation here and now. (One may add that very few people indeed have either the time or the competence to go through the by-now massive body of scientific literature on the subject and to form an objective view of their own. It would seem to follow from this obvious fact that most people, on either side of the controversy, will base their respective positions on something other than rigorously considered scientific judgment.)

These general considerations are relevant in that they lead to the following questions for those who favor this legislation: Do they want to further an overall attitude of unrealistic risk-avoidance in the society? Do they want to stigmatize people and lifestyles that deviate from what is considered proper and wholesome in the elite milieus of the society? More specifically, should public policy and law impose upper-middle-class values and lifestyles on the rest of the population? It seems to me that both realism and democratic values incline one toward negative answers to all three questions. In sum: I don't believe that what American society needs right now is a new version of creeping Prohibition.

The Issue of Government Power

Americans are rightly proud of living in a pluralistic society. In such a society, there will always be conflicts between discrepant values and lifestyles, and evangelism of one's own values and lifestyles is protected by the First Amendment. Also, there will always be conflicting vested interests in such a society, and the idea that these can all be resolved into harmony is utopian and undemocratic. The matter becomes very serious, however, when one ideological party seeks to utilize the powers of government in elevating its own values, lifestyles and interests to monopoly status. In view of the vast powers of the Federal government, this is doubly serious when Federal policy and Federal legislation is involved. In the matter under consideration here, there is "an issue behind the issue" -- the issue of government power. In my opinion, that is the most important issue.

The cultural theme of risk reduction does not necessarily lead to an expansion of government power. After all, there are other ways by which individuals may want to protect themselves from the risks they fear. But, in an age where government is increasingly looked upon as the provider and guarantor of all desiderata in human life, it is only logical that government should be expected to protect people from the risks they want to avoid. In the end, this produces a paradox. Since many of the risks individuals fear are actually incurred as a result of their own actions, government is expected to protect people from themselves -- a curious inversion of classical democratic ideals.

Ivan Illich recently pointed out that the modern state is increasingly taking on the characteristics of the church; it becomes mater et magistra, "mother and teacher," the source of all nurture and all instruction. The "magisterial" or teaching function of the state is particularly evident in the legislation under consideration here. Surely, since the first Surgeon General's report on the alleged perils of smoking, the American public has been bombarded with negative communication about this practice, including the warning notice printed, under penalty of law, on all cigarette packages. It is now alleged that the message has not sufficiently registered and that it ought to be reinforced. One detects here the note of irritation familiar to all teachers whose pupils fail to learn the proper lessons. Such irritation, I suppose, is quite justified when children refuse to learn arithmetic or grammar. In this case, though, it carries the disturbing implication that the American public actually consists of child-like individuals. But what is the evidence for this? As far as I know, the only evidence is that smoking has not declined to the degree that anti-smoking activities would wish. But is it not plausible to argue that adults, with all the information available to them, make their own decisions -- including decisions as to what risks to take?

There is something very disturbing about the notion that government in a democracy has a "magisterial" function. It becomes even more disturbing when government pushes its "teachings" ever more aggressively as people don't take to it in the opinion of the "teachers". A democracy presupposes that its citizens are responsible adults; therefore, when that same government engages in activities that treat citizens as recalcitrant children, they have a right to be offended. The proposed policy of rotational warnings has precisely this character of infantilization: "Now, Johnny, since you haven't learned your lesson, you will write it on the blackboard one hundred times . . . ." Magistra indeed!

The most troublesome aspect here, however, is the implicit threat of an ever-deeper penetration of governmental influences in the private lives of individuals. No reasonable person will deny that, in a modern society, government must protect individuals from risks imposed by others. That is, government denies me the right to impose risks on unwilling or unaware others. It is quite a different matter for government to protect me from risks incurred by me alone as a consequence of my own actions.

An analogous case may be instructive here. When I drive my car, I obviously risk injuring other people. The government, quite rightly, seeks to limit this risk. Thus government examines and licenses drivers, imposes penalties for dangerous driving, mandates various safety devices in cars, and so on. No reasonable person will consider such regulation of behavior a violation of the individual's rights. It is quite another matter, though, when government sets out to protect me against the possible risks of my own driving. This issue surfaced in the debate over involuntary safety devices in cars. Should government not only insist that cars contain properly functioning devices that protect others (such as brakes, lights, and the like), but also devices that, regardless of whether I want them or not, protect me, the driver (such as safety belts without which I cannot start the car, and the like)? There has been strong resistance to the latter extension of government regulation, both in the Congress and in the American public. There has been strong feeling to the effect that it is my business and my business only, if I do not protect myself against the risk of going through the windshield in a collision.

Now, it is not my intention to take a position, one way or another, on the issue of involuntary driver-safety devices in automobiles. But I do want to comment on a concept that has been introduced into that debate and that is relevant to the present controversy as well -- the concept of "social costs". It is one of those ideas which, at first glance, seems eminently sensible -- until one starts thinking it through to its logical consequences. Here is what the concept refers to: To be sure, if I risk serious injury or death as a driver, it is myself that I injure or kill. Nevertheless, there are costs to society. Society (be it directly through the state or indirectly through rising insurance costs) has to bear the burden of my hospitalization and medical treatment; society (again, through state or private insurance mechanisms) has to assume costs resulting from my unemployment or disability; if I'm permanently disabled or die, society has to concern itself with the welfare of my dependents or survivors; and the economy has to adjust to my diminished or terminated productivity. From this perspective, I risk far more than my own life or limb in an automobile accident; rather; I risk imposing burdens on others, to the point of threatening (in whatever measure) the viability of the welfare machinery of the state, the viability of the private insurance system, and even the economic well-being of the society (the latter a sort of offense against the Gross National Product).

When one puts the concept of "social costs" in these terms, one becomes (or should become) pensive, for there is a totalitarian thrust to this logic. After all, what actions of mine do not entail "social costs" in the aforementioned sense? I think one must answer: Very few indeed. Thus, my lazy and self-indulgent lifestyle endangers my health (with all the alleged risks to society enumerated above); my child-raising patterns (so say various experts) risk making neurotics or juvenile delinquents out of my children; my hobby of tinkering with household machinery endangers the livelihood of plumbers and mechanics, not to mention my gardening tastes which are a standing offense to the aesthetic sensibilities of my neighbors -- and so on. In other words, once the concept of "social costs" is accepted as a rationale for government interference with private individual behavior, it is difficult to see just where such interference would stop.

This point is so important that a further analogy is appropriate. In the current controversy, there is constant mention of the alleged economic costs of smoking, this supposedly justifying anti-smoking efforts by government. The manner in which these alleged costs are calculated strikes me as rather fanciful (the figures depend on all sorts of presuppositions, medical as well as economic, that seem questionable) but let that pass here. Let me instead propose that excessive and improper eating constitutes a health hazard. Let me suggest that there is a "cholesterol epidemic" in America. If so, let us look at the individual aspects of this first, and then at the possible policy implications. I habitually overeat, and I eat the wrong things. Cholesterol builds up in my body. In consequence, I greatly increase the risk of suffering a heart attack or a stroke. And, if the aforementioned logic is accepted, I incur this risk not only for myself but for others as well; indeed, I'm a walking bundle of potential "social costs". And now imagine the government interventions that suggest themselves in this case: Rotational anti-cholesterol warnings on all food products; prohibition of advertising of cholesterol-rich foods; intrusive government propaganda on matters of diet; government regulation of all meals served in public places and prohibition of certain types of cuisine (say, Italian restaurants); perhaps tax incentives for health restaurants and food establishments; mandatory physical exercise for workers in government or government-subsidized enterprises (in the manner of Maoist China); and so on. Absurd? Maybe (I hope so). But not if one takes the concept of "social costs" seriously as a guide to legislation and public policy. What is missing so far is an anti-cholesterol movement, arrayed militantly against the "cholesterol interests" and with enough political clout to put items from the above list on the agenda. Given the cultural climate I tried to describe before, this may only be a matter of time.

I feel optimistic about the good sense of Americans in resisting this sort of totalitarian encroachment. They showed good sense in resisting Prohibition, made its enforcement impractical, and finally forced repeal. But, in the meantime, a great deal of damage was done. For one, there was an enormous expansion of organized crime -- perhaps inevitable in retrospect, if government criminalizes a large area of private behavior. It seems, though, that the lessons of Prohibition have been forgotten. Be this as it may, it is very important to be alert to the totalitarian implications of all comparable intrusions of government into the private behavior of individual citizens. Most Americans, across the entire political spectrum, recognize the need for welfare-state measures in modern society (political disagreements are really not about the welfare state as such any longer, but about its extent, its mechanisms and its costs). But the accumulation of risk-reducing demands on government must bring about a quantum leap in the conception of the welfare state -- a leap precisely in the direction of an all-embracing totalitarian understanding of the role of government. If the behavior of individuals is to be politically proscribed or regulated because it is claimed to carry "social costs" in the aggregate, then virtually no area of private life is immune to government intervention.

Again, I would not like to be misunderstood: I am not saying that the legislation under consideration here is totalitarian either in intent or even in its immediate consequences if enacted. But the totalitarian tendencies in modern society are frequently unintended, incremental, "creeping". That is why they must be resisted in their early, seemingly innocuous, stages. Those who favor the measures proposed by this legislation should ask themselves whether they might not recoil from the final outcome. In other words, the road to a coercive society is paved with all sorts of good intentions; each step on the road may look benevolent and sensible, but the destination may be anything but benevolent and sensible.

In conclusion, let me suggest that there is a particular irony to this legislation being proposed at this particular time. Over the last few years, a remarkable bipartisan consensus has developed as to the need to reduce and limit government regulation. It is ironic that, in the name of health, it should now be proposed to increase government regulation in an area of individual private activity. In this particular instance, there is an additional irony, alluded to before in connection with Scandinavian anti-smoking legislation. For a long time, the Scandinavian countries (especially Sweden) were held up as models of the welfare state. Of late, these models have shown themselves to be much less impressive than many people used to think (most importantly, this insight has been growing in Scandinavia itself). It seems to me that we have a very real opportunity in America today to demonstrate that a modern welfare state can be different from the "Swedish model" -- more pluralistic, less costly, less intrusive, working with rather than against private initiative and individual rights. The present case is one of many areas of public policy where, in my view, we can do much better than emulating an obsolete model from abroad.

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Attached: Vita
1977 article

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Associate Professor, then Professor, Graduate Faculty, New School for Social Research, 1963-70
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Major Books:

Invitation to Sociology: A Humanistic Perspective, 1963
The Social Construction of Reality: A Treatise in the Sociology of Knowledge (with Thomas Luckmann), 1966
The Sacred Canopy: Elements of a Sociological Theory of Religion, 1967
A Rumor of Angels: Modern Society and the Rediscovery of the Supernatural, 1969
The Homeless Mind: Modernization and Consciousness, (with Brigitte Berger and Hansfried Kellner), 1973
Pyramids of Sacrifice: Political Ethics and Social Change, 1975
The Heretical Imperative: Contemporary Possibilities of Religious Affirmation, 1979
Sociology Reinterpreted: An Essay on Method and Vocation, 1981

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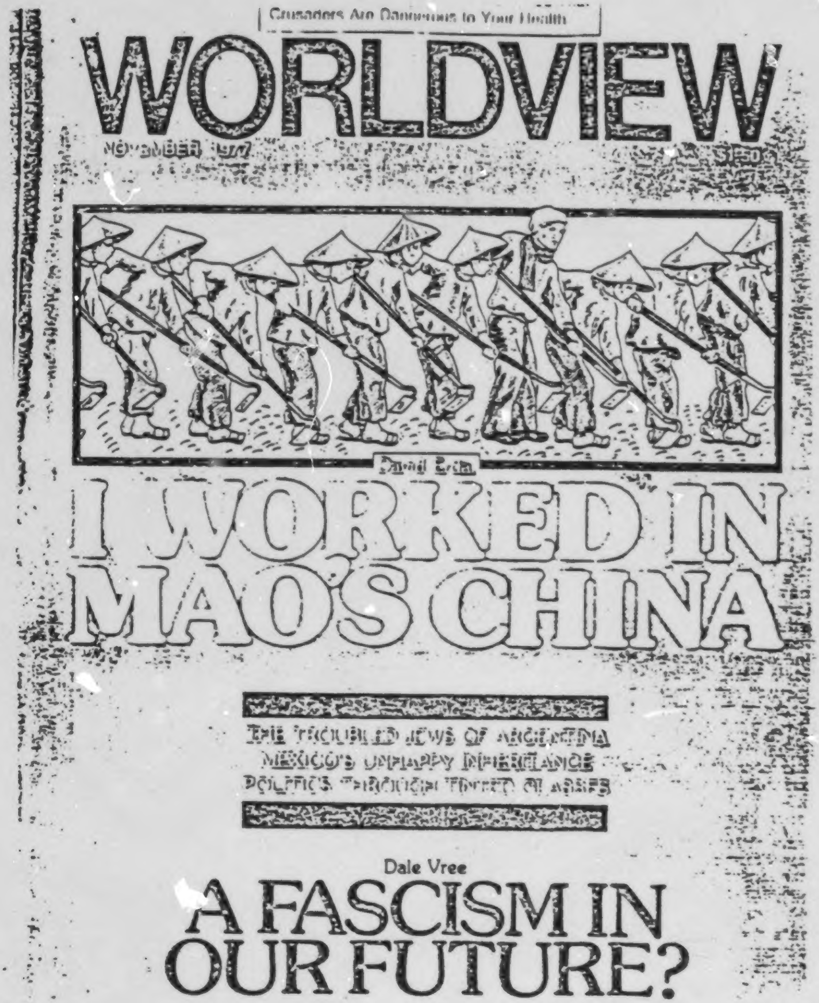

Crusaders Are Dangerous to Your Health

WORLDVIEW

NOVEMBER 1977 $1.50

David [illegible]

I WORKED IN MAO'S CHINA

THE TROUBLED JEWS OF ARGENTINA
MEXICO'S UNHAPPY INHERITANCE
POLITICS THROUGH TINTED GLASSES

Dale Vree

A FASCISM IN OUR FUTURE?

Observation

Gilgamesh on the Washington Shuttle

Peter L. Berger

The antismoking forces are gaining in political muscle all over the country. Laws and municipal ordinances are passed to prohibit smoking in every sort of public place and, where that is not (or not yet) feasible, to segregate smokers in ever-smaller areas. There is pressure to further restrict advertising for cigarettes and to abolish federal subsidies for tobacco growers. But what is most interesting on the level of everyday life is the aggressiveness and the moral fervor with which this campaign is being conducted. There are segments of the American population in which the hostility to smoking and to smokers has taken on the quality of a crusade. And if we have learned anything from the politics of this century, it is to pay nervous attention to any new crusades appearing on the scene.

I freely confess that I'm not a disinterested observer: I smoke. Also I'm given to political paranoia: I can foresee a time in the near future when smoking will be prohibited everywhere except by consenting adults in the privacy of their bedroom (and, to be honest, I don't trust the antismokers to stop short of the bedroom for very long either). Or, alternatively, I can see myself puffing my cigarillos in a fenced-off area near the toilets in my favorite restaurant, or slinking through the streets with a scarlet "S" pinned on my lapel, or having to make biweekly visits to a social worker to have my permit renewed (you see, I refused group therapy). Let these visions not be taken lightly: I have developed a *wissenschaftliche* respect for my paranoias; most of them have turned out to be predictions, at least when it comes to political reality.

Some stipulations, to be sure, are in order. Undoubtedly there are places where nonsmokers have long been annoyed by the proximity of smokers. Undoubtedly there have been thoughtless smokers who have caused discomfort, sometimes even real suffering, to others. No fair-minded person, even one who smokes twenty cigarillos a day, will object to rules that protect those caused discomfort or pain by people blowing smoke at them. But the current campaign goes far beyond the establishment of such rules. More important psychologically, the aggressiveness of the campaign cannot be explained by such rational grievances. Also, of course, there is evidence to the effect that smoking is unhealthy (though, let it be added, the evidence is less conclusive than the antismoking crusaders would have us believe).

But, once again, the thrust and the mood of the campaign make it implausible to seek motives in concern for smokers' health. Not for a moment do I believe that these people want to protect me from emphysema. As a matter of fact I believe they would be quite delighted if I got emphysema—or worse—as just punishment for my wicked habit. It's sort of the way I assess the motives of Jehovah's Witnesses: They stand on the street corner offering me a copy of *Awake!* because it's their duty to do so. But they *know* that come the day of reckoning very few will be saved—and, the way they look at me, I know that they don't really mind my passing by my chance of joining that élite. In the business of salvation it's long been true that "the fewer, the merrier," and one doesn't have to read Jonathan Edwards to know that the well-deserved suffering of the damned is one of the pleasures of heaven.

I'm digressing. Back to *Wissenschaft*: Sex is in; smoking is out. If you don't believe me read the personals columns in the left-wing press (much to be recommended generally as a depth-probe into the consciousness of the New Class; by their libidos thou shalt know them) and count the number of times that nonsmoking is listed as a trait wanted in sought-after sex partners. It's come to this: "Trim college professor, late 30's, seeks liberated female for complementary relationship [that means he's married], Tuesday and Thursday afternoons, NYC or Connecticut —*nonsmoker*." "Westchester County couple, happily married, seeks young man or woman, early 20's, for mutually satisfactory explorations in new life-styles—*nonsmokers*." "Hispanic male, currently incarcerated, invites correspondence from submissive females, race immaterial—*nonsmokers*...."

Recently (as the result of a lawsuit, I believe) Eastern Airlines substantially decreased the space allowed to smokers on its planes. The new restriction also applies to the New York-Washington shuttle, a conveyance vital to the existance of politicians, lobbyists, agents of the

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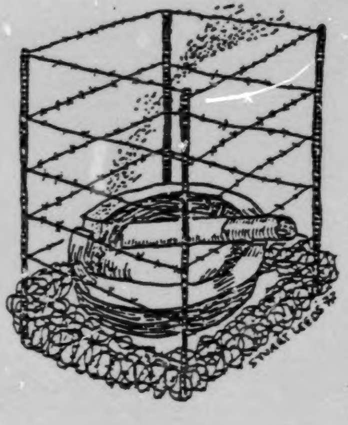
South Korean Government, individuals who carry a hundred thousand dollars in attaché cases, and other people with nervous-making life-styles, mostly males, mostly smokers. But never mind: Justice is blind.

So the other day I was on the shuttle, joining the desperate rush toward the new Jim Crow section in the back of the plane near the toilets, where they keep the oxygen equipment and the old copies of *Business Week*. I lost the race to the last smoker's seat to a fat young man in a seersucker suit, with ten ballpoint pens stuck in his shirt pocket and clutching *two* attaché cases (an agent for both the South Korean *and* the North Korean governments?). Consequently I was forced to sit in rigid abstinence in the enormously enlarged nonsmokers' section (the man next to me, smiling sheepishly, smoked *furtively*—I was tempted to emulate him, but I'm a law-abiding citizen; also I'm given to anxieties, and the stewardess—pardon, the flight attendant—bore a striking resemblance to Bella Abzug...). I suffered (it so happens that I'm also given to fear of flying—Erica Jong hasn't done a thing for *me*—but that's another story). As everyone knows, suffering is conducive to insight. And so somewhere over Chesapeake Bay (the plane suddenly lurched and the engines didn't sound quite right for a moment there) I had what my teacher Alfred Schutz used to call an "*aha* experience." I think I know now what is going on in this department of our culture: *Antismoking is the new anti-Semitism. And, like the old anti-Semitism, it has to do with the quest for immortality.*

I recognize that, at first reading, these propositions may lack plausibility. I suggest that the propositions be scrutinized by following a very simple analytic procedure (*Wissenschaft* again): The antismoking attitude is *one item* in the consciousness of a specific group of people; it should be seen in relation to *other items* in that consciousness. At that point, I contend, my propositions gain plausibility. But first: Who are these people? That question is easily answered: We are talking about the aforementioned New Class—the people who consider themselves intellectuals but who are better described as that new segment of the upper middle class that derives its living (and ipso facto its material and ideological interests) from the so-called knowledge industry.

Daniel Bell and Irving Kristol have given us the basic sociological understanding of this class. It is a minority in the American population but, because of its relation to the education and mass communications systems, a very influential one. It is both the matrix and the principal clientele of most of our recent movements of cultural innovation, from the "new consciousness" to the "new politics." Many recent events on the American scene make much better sense if one sees them as part of a class struggle (in quite classical Marxist terms) between this New Class and the old upper middle class (Kristol calls the latter, *tout court*, the business class). The outcome of this class struggle has not yet been decided, but there is little doubt that both the cultural and the political power of the New Class has been on the rise.

Not all elements of the consciousness of this class are relevant to the antismoking issue. But a surprising number are. These are all related to an overwhelming sense of corruption and pollution from which the society is to be cleansed. Ask this simple question. What are *other causes* to which the antismokers are likely to be attracted? There is, probably most important of all, the various causes espoused by the environmentalist movement. It began with Ralph Nader's war against the automobile. It finds its foremost expression today in the campaign against nuclear energy. Modern industrialism (and, in the consciousness of the New Class, especially its capitalist version) is perceived as a gigantic death-machine, destroying the earth, poisoning the atmosphere, spreading every sort of plague. Then there are the causes related to the various health movements. Commercially produced foods are supposed to poison us, and the only salvation is in the return to some form of organic nutrition. The drugs produced by the pharmaceutical industry (with the possible exception of hallucinogenic ones) are also death-dealing, and the medical establishment has been an accomplice in this conspiracy to destroy us (Ivan Illich—perhaps unintentionally—has helped to legitimate this particular hypochondria). And then there is the strong affinity of this same class for every conceivable type of therapy, from the conventional services of the psychoanalyst to the latest products of the California personal-liberation industry.



Other elements could be mentioned, but these three will do for the present purpose. What do they have in common? Hypochondria is as good a term as any. But one could also put it differently: What is said here is that all of us would lead long and

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healthy lives, *if only this or that particular source of pollution were removed*.

Thus the cultural scene is rampant with utopias of health: Get rid of the automobile, ban nuclear energy—and we would all live in bucolic idylls, riding our bicycles along crystalline brooks. Eat nothing but food grown on natural manure—and you'll never get ill. Follow this or that therapeutic treatment and you will never be anxious again, never frustrated, never defeated. Is it too fanciful to push these promises to their final limit? I don't think so. For here is the finally implied promise: *Do these things—and you will live forever*. Only if one understands this can one understand the intense hostility to those (be they individuals or "the system") who are believed to stand in the way of this vision of eternal health: *They*—the carmakers, the advocates of nuclear energy, the makers of nonorganic breakfast cereals, those who stop me from having all the orgasms I need—and of course the smokers—*they* are the enemies of life, the purveyors of death, the one great obstacle between me and the redeemed state.

It is not necessary to deny all empirical complaints of these movements in order to make this analysis. Thus, for example, there are very real threats to life in the diffusion of nuclear energy, threats that ought to be taken very seriously in the public debate. In other words, I'm *not* saying that there are no rational arguments on the side of the Clamshell Alliance; I *am* saying that these rational arguments are insufficient to explain the phenomenon of the current antinuclear movement. Indeed, almost every cause has some kernel of truth, but to understand the energizing passions behind a cause the validity of its empirical claims is often beside the point. This is unfortunate, to be sure, but it cannot be helped (those who have not learned this lesson from Marx may learn it from Vilfredo Pareto).

Jean-Paul Sartre, in his book *Anti-Semite and Jew* (incidentally, his last intelligent comment on any politically relevant issue), interpreted modern anti-Semitism as a flight from the human condition. It was a profound and profoundly correct interpretation. For the anti-Semite it is the Jew who is the enemy of life, the corrupting and polluting force eating away at the health of the society. Therefore in battling the Jew the anti-Semite is at the same time struggling for his own redemption, and the victory over the Jew is ipso facto a victory of life over death. This vision, Sartre tells us, is only possible by denying some fundamental and ineradicable elements of the condition of being human. For to be human means to live in an imperfect world, with all the anxieties and ills that go with this. Most important of all, to be human means to be mortal. Thus anti-Semitism, in the final analysis, is a flight from mortality and (in the mode that Sartre called "bad faith") a denial of mortality.

Let me make clear that I'm *not* suggesting a *moral* equivalence between anti-Semitism and these contemporary forms of utopianism. Anti-Semitism, even in its less virulent forms, is a phenomenon sui generis, with a moral perversity all its own that should not be trivialized by throwing it into the same category with a lot of other phenomena. Nevertheless it is very useful to understand the common elements it shares with other turbulences of contemporary consciousness. Nor should we overlook the fact that a new variety of anti-Semitism (still generally camouflaged as "anti-Zionism") has found a response precisely in the utopian milieu being discussed here.

One significant fact must still be added to the argument: The group that is given to these utopias is almost certainly the most secularized sector of the American population. In other words these are people who have been deprived (or, if you will, who have deprived themselves) of the consolations of religion in the face of mortality and all the other imperfections of the human condition. It is not surprising, then, that the flight from mortality takes on a rather frantic character in this particular ambience. In the absence of God the prospect of death becomes much harder to deal with. There is, of course, the great possibility of stoicism, but very few are capable of that. (Perhaps Sigmund Freud was the last great stoic. Certainly not Sartre, who got hooked on the "bad faith" of revolution.)

And thus it is with us again, that age-old quest for the secret of immortality. Long, long ago Gilgamesh looked for it in his journeys to find the Land of the Living. For a moment he thought he had found the secret in a plant that grew on a faraway shore, and he exclaimed: "This plant is a plant apart, whereby a man may regain his life's breath....Its name shall be 'Man Becomes Young in Old Age.' I myself shall eat it, and thus return to the state of my youth." Then a serpent came out of the water and carried off the plant and (according to the Akkadian version of the Gilgamesh story) "Gilgamesh sat down by the water and wept, his tears running down over his face." One may say that the whole drama of biblical religion is one long effort to wipe away these tears over the anguish of human finitude—but the effort is not an easy one, and its fundamental presupposition is an acceptance of reality and a turning away from the illusions of false promises. It was another serpent, speaking of another plant, who promised: "*You will not die*....When you eat of it your eyes will be opened, and you will be like God."

But let me return from the ancient Near East to the Washington shuttle. We are all sinners. None of us are all that good at coping with the human condition, none of us have sufficient faith. (If I did I'm sure I wouldn't smoke. For what smoking is finally all about is a profound impatience before the mystery of time, and this too is a lack of trust in God who is the Lord of time. But that is yet another story.) All of us are fugitives from mortality. Perhaps we can acquire a measure of tolerance from this understanding: Tolerance of each other—which precludes crusades. And tolerance of the imperfections of the world—which frees us from the illusion of utopianism.

Do you happen to have a light, Ms. Gilgamesh? Thank you. No, I'm not sure either where this plane is going....

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STATEMENT

RODGER L. BICK, M.D.

My name is Rodger L. Bick; I am the medical director of the San Joaquin Hematology & Oncology Medical Group. I am also an assistant professor of medicine, specializing in hematology and medical oncology, at the School of Medicine of the UCLA Center for the Health Sciences in Los Angeles. I am on the clinical faculty for hematology and oncology of the Wadsworth V.A. Hospital in Los Angeles, and hold adjunct teaching positions at the Wayne State University Specialized Center for Thrombosis Research in Detroit and at the Wesley Medical Center and University of Kansas Medical School in Wichita.

I received my medical degree from the University of California at Irvine School of Medicine in 1970. In 1973 and 1974, I was the Director of the Hemostasis/Thrombosis Research Laboratory, Chief of Hematology/Medical Oncology, and Director of Medical Education at the Kern County General Hospital in Bakersfield, California. From 1974 till 1977, I worked for the Bay Area Hematology Oncology Medical Group and Bay Area Hematology Oncology Clinical and Research Laboratories in Santa Monica. From 1980 to date, I have been an associate professor of Allied Health Professions and of Nursing and Health Sciences at California State University in Bakersfield.

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I am a member of numerous professional societies, including the International Society on Thrombosis and Haemostasis and the American Society of Hematology, and serve as editor for several journals in my field. I have written over 100 published articles on various aspects of the field of hematology.

I have long been interested in the subject of blood coagulation and its possible relationship to heart attacks and cardiopulmonary disease in general. Recent reports have appeared in the literature describing alleged isolation and biological properties of certain brown pigments from cured tobacco leaf, cigarette smoke condensates, and saline extracts of smoke. These pigments have been generally referred to as "tobacco glycoprotein" (TGP). Becker and co-workers have reported that TGP produces allergic skin reactions, induces the formation of antibodies, and activates early clotting factors. Based upon these rather simple experiments, with respect to early blood clotting factors, Becker and co-workers have drawn far reaching conclusions that TGP therefore must also activate the remainder of the clotting system, clot lysing system, and complement system. However, there is no evidence for this sequence of events in either the Becker articles or in subsequent work. In spite of this lack of evidence, these authors have hypothesized that these possible sequential pathological events represent the pathophysiological link between cigarette smoking and cardiopulmonary disease which has been reported.

To investigate more carefully these claims by Becker et al., our group, in collaboration with Dr. Stedman, extracted TGP by the previous published methods of Becker and co-workers. Our group found that the final product obtained by the isolation procedure of Becker contains and consists primarily of a contaminant that is introduced by the methods of separation and appears to be a side product of the photo-polymerization reaction used to form the gels for separation. (Relatives of this contaminant, polymethylmethacrylates, are widely used in orthopedic and dental surgery and traces of these compounds have been implicated in allergic reactions and clot formation.) When we subjected the contaminant to the aforementioned coagulation, clot lysing, complement, and kinin generation assay systems, we found that the contaminant complexed with small amounts of an early coagulation Factor, Factor XII, giving the superficial and completely erroneous impression that activation was occurring. However, it was found to have no activity on thrombogenic assays currently used by the Bureau of Biologics for detecting activation of the blood clotting system. Nor was there any activation of numerous of the early coagulation Factors tested. Likewise, there was no activation of the fibrinolytic system, no effect on the complement system, and no effect on the kinin generating system. Further, we found that there was no effect of this contaminant on the normal clot inhibiting proteins found in human blood.

Our studies strongly suggest that TGP isolated by the general published procedure of Becker and co-workers consists of a contaminant which is introduced during the separation process and contributes to, or more likely, is entirely responsible for the biological activites noted by Becker and co-workers.

The presence or absence of a substance in tobacco smoke with biological properties supposedly effecting the coagulation, clot lysing, complement, or kinin system remains controversial and certainly unproven. It is obviously of importance to examine the significance, if any, of tobacco glycoprotein; however, a good deal of controversy does exist and it has been impossible to duplicate previous findings. In this one instance it appears that erroneous and superficial observations have led to far reaching hypotheses which cannot be confirmed by carefully performed studies. This author's plea would be for adherence to confirmed scientific fact, rather than emotion, in attempting to study and delineate the biological effects, if any, of cigarette smoking on the blood coagulation complement or kinin systems.

My own research and review of the scientific literature lead me to the conclusion that whether and how smoking might cause heart attacks or cardiopulmonary disease remain open questions.

CURRICULUM VITAE: RODGER L. BICK, M.D.

Born: San Francisco, California 5/21/42
Marital Status: Married

EDUCATION:

Primary: Madera Grammar School, Madera, California
Secondary: Madera Union High School, Madera, California
Undergraduate: University of California at Berkeley [illegible]
Minor: Physics.
Graduate: University of California at Irvine, School of Medicine, M.D. Degree, June, 1970.

TRAINING:

Internship: Straight Medicine, Kern County General Hospital July 1970 - June 1971, Bakersfield, California
Residency: Internal Medicine, Kern County General Hospital, July 1971 - June 1972, Bakersfield, California
Fellowship: Hematology/Medical Oncology, Department of Medicine School of Medicine, UCLA Center for the Health Sciences, Los Angeles, California

Coagulation Hematology Fellowship: Research Division Hyland Laboratories, Affil. UCI School of Medicine, Costa Mesa, California, June 1966 - September 1966.

Coagulation Hematology Fellowship: Research Division Hyland Laboratories, Affil. UCI School of Medicine, Costa Mesa, California, June 1967 - September 1967.

HONORS:

Dean's List and Honors Student, University of California at Berkeley, [illegible], 1965.
California College Honors Society.
Honors Day Award for Outstanding Research, University of California at Irvine, School of Medicine, 1967.
Honors Day Award for Outstanding Research, University of California at Irvine, School of Medicine, 1968.
[illegible] P. Carrull Award: To Senior Medical Student contributing most to medical research during medical school tenure, June 1970.

APPOINTMENTS:

Director Hemostasis/Thrombosis Research Laboratory, Kern County General Hospital, Bakersfield, California, July 1973 - May 1974.
Chief: Hematology/Medical Oncology, Kern County General Hospital, Bakersfield, California, July 1973 - May 1974.
Director of Medical Education: Kern County General Hospital, Bakersfield, California, July 1973 - May 1974.

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CURRICULUM VITAE: RODGER L. BICK, M.D.

APPOINTMENTS (cont.):

Member: Cancer Committee, St. John's Hospital, Santa Monica, California
Member: Research Committee, Santa Monica Hospital, Santa Monica, California.
Member: Tumor Board, Santa Monica Hospital, Santa Monica, California
Member: Cancer Committee, Santa Monica Hospital, Santa Monica, California
Consultant: Nigeria Hematology Center, Lagos, Nigeria.
Founding Director: Oncology Unit, Santa Monica Hospital, Santa Monica, CA
Director: National Hemostasis Thrombosis Seminars & Workshops, Commission on Continuing Medical Education, American Society of Clinical Pathologists
Medical Staff: UCLA Center for the Health Sciences, Los Angeles, CA
Medical Staff: Santa Monica Hospital and Medical Center, Santa Monica, CA
Medical Staff: St. John's Hospital and Health Center, Santa Monica, CA
Attending Physician: (Hematology-Oncology): Wadsworth Veterans Hospital, Los Angeles, California
Medical Staff: Greater Bakersfield Memorial Hospital, Bakersfield, CA
Medical Staff: San Joaquin Community Hospital, Bakersfield, CA
Medical Staff: Mercy Hospital, Bakersfield, California
Chairman: Cancer Committee, San Joaquin Community Hospital, Bakersfield, CA
Chairman: Tumor Board, San Joaquin Community Hospital, Bakersfield, CA
Founding Director: Oncology Unit, San Joaquin Community Hospital, Bakersfield, California
Clinical Investigator: UCB Pharmaceuticals, Brussels, Belgium.
Clinical Investigator: American National Red Cross, Bethesda, Maryland.
Co-Investigator: AT-III Project, Bureau of Biologics, Food and Drug Administration.
Consultant: Council for Tobacco Research
Research and Grant Peer Review Committee: American Heart Association
Clinical Investigator: Kabi Group
Clinical Investigator: American DADE
Clinical Investigator: Coulter Electronics
Member: Board of Editors, Educational Materials Advisory Committee, American Society of Clinical Pathologists.

FACULTY APPOINTMENTS:

Assistant Professor of Medicine (Hematology/Medical Oncology), Department of Medicine, School of Medicine, UCLA Center for the Health Sciences, Los Angeles, California
Clinical Faculty (Hematology/Oncology) Wadsworth V.A. Hospital, Los Angeles, California
Associate Professor of Allied Health Professions, California State University Bakersfield, California 1980-81
Clinical Associate Professor of Nursing and Health Sciences, California State University, Bakersfield, CA 1981-82
Adjunct Associate Professor of Medicine/Physiology, Specialized Center for Thrombosis Research, Wayne State University, School of Medicine, Detroit, Michigan (Trainee to rotate to Bakersfield and I lecture there)
Adjunct Clinical Faculty: Wesley Medical Center and University of Kansas Medical School, Wichita, Kansas (Pathology Research Rotation under Rodger L. Bick, M.D., in Bakersfield, California for Hemostasis/Thrombosis).

PRIVATE PRACTICE:

Bay Area Hematology Oncology Medical Group and Bay Area Hematology Oncology Clinical and Research Laboratories, Santa Monica, California, 1974 - 1977.
Medical Director: California Coagulation Laboratories, Inc., Bakersfield, CA.

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PRIVATE PRACTICE (cont.):

San Joaquin Hematology Oncology Medical Group, Inc., Bakersfield, California
November 1977 to present.

PATENTS PROCURED:

A. A Method for the Large Scale Removal of HAA from Plasma; Bick, RL.
B. A Method for the Large Scale Fractionation of Low Protein, Low Fibrinogen, High Potency Factor VIII Concentrate, Bick, RL, and Fekete, LF.
C. A Method for the Large Scale Fractionation of Antithrombin III, Bick, RL and Fekete, LF.

PUBLICATIONS:

Co-Editor for Seminars in Thrombosis and Hemostasis, Stratton Intercontinental Medical Book Corporation, New York.

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BOOKS/CHAPTERS/MANUALS:

Bick, RL: Editor and Main Author
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Guest Editor: Seminars in Hemostasis and Thrombosis, October 1976.
Alterations of hemostasis associated with cardiopulmonary bypass; pathophysiology, prevention, diagnosis and management. R.L. Bick,
Chapter 2: Laboratory modalities for assessing hemostasis during cardiopulmonary bypass. Bick, RL and Fekete, LF.

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Chicago, Ill. 1976 (300 pages)

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Current Concepts of Hemostasis and Thrombosis: Clinical and Laboratory Evaluation
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CRC Press, Boca Raton, FL 1980.

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Basic Concepts of Hemostasis and Thrombosis, Murano, G. and Bick, RL (eds.)
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Bick, RL:
Hypercoagulability and thrombosis, Chapt. 13 in
Basic Concepts of Hemostasis and Thrombosis, Murano, G. and Bick, RL (eds.)
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Bick, RL:
Anticoagulant and antiplatelet therapy. Chapt. 14 in
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Murano, G. and Bick, RL:
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Bick, RL:
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Bick, RL:
Hemorrhagic problems associated with cardiac surgery. In Preparation, Chapt. in
Textbook of Hemostasis and Thrombosis, Ratnoff, OD, and Forbes (eds.)
Grune & Stratton, New York 1982.

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BOOKS/CHAPTERS/MANUALS (cont.):

Bick, RL:
Hemostasis in transplantation surgery. In Preparation - for
Textbook of Hemostasis and Thrombosis, Ratnoff, OD and Forbes (eds.)
Grune & Stratton, New York 1982.

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Bick, RL, Shanbrom, E, Bishop, R:
Fibrinolytic abnormalities in myeloproliferative disorders.
Clin. Res. 21:264, 1970

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ABSTRACTS (cont.):

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Hemorrhagic syndromes associated with cardiopulmonary bypass
pathogenesis, diagnosis, and management.
Trans. XII World Congress Int. Cardiovas. Soc. p. 7, 1975

Bick, RL:
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Thromb. Diath. Haemorrh. 34:875, 1975.

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Bick, RL, Schmalhorst, WR, Arbegast, N:
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Am. J. Clin. Path. 63:588, 1975

Bick, RL:
Fibrinolytic enzymes in metastatic cancer
Am. J. Clin. Path. 65:266, 1976

Bick, RL, Fekete, LF, Wilson, WL:
Treatment of disseminated intravascular coagulation with antithrombin III.
Trans. Am. Soc. Hematol. 167, 1976.

Bick, RL, Klein, CA, Fekete, LF, Wilson, WL:
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Fekete, LF, Wilson, WL, Bick, RL:
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high potency factor VIII concentrate.
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Bick, RL, Dukes, ML, Fekete, LF, and Wilson WL:
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Philadelphia, 1977.

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ABSTRACTS (cont.):

Bick, RL, Dukes, ML, Fekete, LF:
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Bick, RL, Fekete, LF, Wilson, WL:
Platelet adhesion in clinical medicine.
Trans. First Florence Conference on Hemostasis and Thrombosis
Florence, Italy, 1977.

Bick, RL, Arbegast, NR, Fekete, LF, and Wilson, WL:
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Am. J. Clin. Path. 68:93, 1977

Bick, RL, Dukes, ML, Wilson, WL, Fekete, LF:
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Bick, RL, and Fekete, LF:
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Bick, RL, and Wilson, WL:
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Bick, RL, and Wilson, WL:
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The development and evaluation of a new coagulation standard for protimes, PTT's and all coagulation factor assays.
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Bick, RL, Fekete, LF:
Alterations of hemostasis during cardiopulmonary bypass: A comparison between membrane and bubble oxygenators.
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Bick, RL, and Fekete, LF:
Hereditary hemorrhagic telangiectasia and associated thrombohemorrhagic defects.
Blood, 54:745, 1979.

Bick, RL, and Skondia, V:
Piracetam: results of a preliminary in vivo trial as a platelet suppressant.
Blood, 54:635, 1979.

Bick, RL, Fekete, LF, Dukes, ML, Gaal, PG, Smith, R:
Hereditary hemorrhagic telangiectasia and associated thrombohemorrhagic defects.
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Alterations of hemostasis during cardiopulmonary bypass (CPB): A comparison between membrane and bubble oxygenators.
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ABSTRACTS (cont.):

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Bick, RL:
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Bick, RL, Skondia, V, and Fritch, D:
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Clinical Research, Vol. 29, No. 2, 400 A, 1981.

Bick, RL:
Hereditary hemorrhagic telangiectasia and disseminated intravascular coagulation: a new clinical syndrome
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Bick, RL, Fareed, J, and Skondia, V:
Piracetam: a new platelet suppressing drug.
Thromb. Haemostas. 46:67, 1981.

Bick, RL, Nix, MK, and Skondia, V:
The effect of piracetam in preventing recurrent deep venous thrombosis.
Thrombos. Haemostas. 46:91, 1981.

Bick, RL, Arbegast, MR:
A comparison of chromogenic, fluorogenic, and fibrinogen substrate assays for determination of plasma heparin.
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Studies related to tobacco glycoprotein: a claimed activator of coagulation, fibrinolysis, complement, kinin, and a claimed allergen.
Thrombos. Haemostas. 46:231, 1981.

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ABSTRACTS: (cont.)

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Bick, RL, and Faulstick, D:
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Bick, RL, Arbegast, NR, and Schmalhorst, WR:
Alterations of hemostasis during cardiopulmonary bypass (CPB): a comparison between membrane and bubble oxygenators.
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Bick, RL:
Disseminated intravascular coagulation: a clinical/laboratory correlation in 48 patients.
Thrombos. Haemostas. 46:395, 1981.

Bick, RL, Skondia, V, and Fritch, D:
Hypercoagulability in diabetes and its correction by an antiplatelet drug: Piracetam.
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Bick, RL and McClain, BJ:
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Am. J. Clin. Path. 77:238, 1982.

Bick, RL:
Disseminated intravascular coagulation: a clinical laboratory correlation in 48 patients.
Am. J. Clin. Path. 77:244, 1982.

LETTERS PUBLISHED:

Bick, RL Anhalt, J:
Needle malaria
N. Eng. J. Med. p. 856, April 1971.

Bick, RL, Anhalt, J:
Malaria in drug addicts
J.A.M.A. 216:1076, 1971

Bick, RL:
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Bick, RL:
Antithrombin III and factor VIII in patients with neoplasms.
Am. J. Clin. Path. Vol. 69, No. 2, 1978.

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CURRICULUM VITAE: RODGER L. BICK, M.D.

SCIENTIFIC PRESENTATIONS/INVITED SPEAKER/WORKSHOP DIRECTOR:

The effects of pinealectomy and environmental lighting on the gonadal, thyroid, and total body weight of female Long-Evans Rats.
Presented to Am. Assoc. Anatomist, New Orleans, 1968.

Simplified measurements of fibrinolytic potential in myocardial infarction.
Presented to Am. Med. Association Multidiscipline Forum.

Fibrinolytic activity in various clinical states.
Presented to Am. Soc. Clin. Path., Las Vegas, Nev. 1971.

Australia Antigen: A comparison of three detection methods in a population of high risk for hepatitis.

Hyperfibrinolysis in cirrhosis.
Presented to Am. Coll. Physicians, Regional Meeting, Los Angeles, CA., 1972.

The special problem of disseminated intravascular coagulation in malignancy
Presented to Am. Cancer Society, Regional Meeting, 1972.

Mechanisms of anemia in malignancy.
Presented to American Cancer Society, Regional Meeting, 1972.

Prothrombin complex concentrate: use to control the hemorrhagic diathesis of chronic liver disease.
Presented to Am. College of Physicians, Regional Meeting, Palm Springs, CA., 1973.

Heparin therapy for disseminated intravascular coagulation
Presented to UCLA 3rd Annual Anesthesia Postgraduate Seminar, UCLA, 1973.

A systemic approach to the diagnosis of bleeding disorders.
Presented for UCLA Postgraduate Seminar in Internal Medicine:
Current Trends in Internal Medicine, January, 1974.

A systemic approach to the diagnosis of anemias.
Presented to UCLA Int. Medicine Seminar.
Current Trends in Internal Medicine, January, 1974.

Current therapy for leukemias and lymphomas
Presented for UCLA Internal Medicine Postgraduate Seminar;
Current Trends in Internal Medicine, January, 1974

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SCIENTIFIC PRESENTATIONS/INVITED SPEAKER/WORKSHOP DIRECTOR:

Therapy for metastatic solid tumors.
Presented for UCLA Postgraduate Internal Medicine Seminar:
Current Trends in Internal Medicine, January, 1974.

Malignant thymoma: A review of 20 cases.
Presented to American College of Physicians, New York, 1974.

Hemostatic Defects Induced by Cardiopulmonary Bypass.
Presented to International College of Angiology, Montreal, 1974.

Hemostatic defects induced by cardiopulmonary bypass.
Presented to the American Association of Clinical Pathologists,
Washington, D.C., 1974.

Control of bleeding associated with cardiopulmonary bypass: Panel member.
National Meeting of the Southern Thoracic Society, Williamsburg, Virginia, 1974.

Special Symposium Participant: "Side Effects of Antihemophilia Concentrates".
World Federation for Hemophilia, Helsinki, Finland, 1975.

Hemorrhagic Syndromes Associated with Cardiopulmonary Bypass:
Pathogenesis, Diagnosis, and Management. International Cardiovascular
Society (yearly meeting), Edinburgh, Scotland, 1975.

Bleeding Associated with Cardiopulmonary Bypass. Cardiology Grand Rounds,
UCLA, 1975.

Bleeding Problems in the O.R. Special UCLA Lecture series; Department of
Surgery, Kern County General Hospital, Bakersfield, California, 1975

A Systematic Approach to the Diagnosis of Bleeding Disorders. UCLA,
Santa Monica Hospital II Annual Family Practice Refresher Postgraduate
Course, UCLA, 1975.

Program and Workshop Director: Recent Concepts and Evaluation of Disorders
of Hemostasis, American Society of Clinical Pathologists, 1975.

Platelet Adhesion, Bleeding Times, and Aspirin
Presented to the American Society of Clinical Pathologists, Chicago, Ill.
1975.

Prothroubin Complex Concentrates and Disseminated Intravascular Coagulation.
Presented to the World Federation of Hemophilia, Helsinki, Finland, 1975.

Recent Concepts and Evaluation of Disorders of Hemostasis and Thrombosis.
American Society of Clinical Pathology National Workshop, September, 1975.

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SCIENTIFIC PRESENTATIONS/INVITED SPEAKER/WORKSHOP DIRECTOR (cont.)

Fibrinolytic Enzymes in Metastatic Cancer.
Presented to the American Society of Clinical Pathologists, National Meeting, Chicago, Ill., September, 1975.

Recent Advances in Laboratory Aspects of Internal Medicine: von Willebrand's Disease, Disseminated Intravascular Coagulation and Primary Hyperfibrino(geno)-lytic Syndromes. Palm Springs, CA., October, 1975.

Hemorrhagic Syndromes Associated with Cardiopulmonary Bypass. An approach to Differential Diagnosis and Management.
Presented to the American Thoracic Society, 1975.

Director: Modern Concepts and Evaluation of Thrombosis and Hemostasis Coagulation Seminar.
American Society of Clinical Pathologists, National Meeting, Dallas, Texas, 1976.

Director: Special Diagnostic Problems in Hemostasis and Thrombosis: Advanced Coagulation Seminar.
American Society of Clinical Pathologists, National Meeting, Dallas, Texas, 1976.

Modern Concepts and Evaluation of Hemostasis and Thrombosis: Two Day Seminar, UC at Santa Barbara, California, 1976.

Director: Recent Concepts and Evaluation of Hemostasis and Thrombosis.
American Society of Clinical Pathologists, Regional Center, Long Beach, California, 1976.

Director: Recent Concepts and Evaluation of Hemostasis and Thrombosis.
American Society of Clinical Pathologists, National Meeting, Los Angeles, California, 1976.

Director: Difficult Diagnostic Problems in Hemostasis and Thrombosis (Advanced Seminar)
American Society of Clinical Pathologists, National Meeting, Los Angeles, California, 1976.

Director: Recent Concepts and Evaluation of Hemostasis and Thrombosis, American Society of Clinical Pathologists, Regional Center, Chicago, Illinois, November, 1976.

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Current Concepts of Hemostasis; Clinical and Laboratory Evaluations of Thrombohemorrhagic Phenomena; Director
American Society of Clinical Pathologists, Commission on Medical Laboratory Personnel Regional Education Program, Los Angeles, 1977

Modern Concepts and Evaluation of Hemostasis and Thrombosis; Director
American Society of Clinical Pathologists, Fall Meeting, Las Vegas, Nevada 1977

Disseminated Intravascular Coagulation and Related Syndromes; Director
American Society of Clinical Pathologists, Fall Meeting, Las Vegas, Nevada, 1977

Difficult Diagnostic Problems in Hemostasis and Thrombosis; Director
American Society of Clinical Pathologists, Fall Meeting, Las Vegas, Nevada, 1977

Recent Concepts and Developments in Evaluating Disorders of Hemostasis and Thrombosis
AMA/CMA, Palm Springs, 1978

Second Annual Southern California Blood Clotting Symposium
American Society of Clinical Pathologists, Santa Monica, 1978

Modern Concepts and Evaluation of Hemostasis and Thrombosis; Director
American Society of Clinical Pathologists, March, Dallas, 1978

Disseminated Intravascular Coagulation and Related Syndromes; Director
American Society of Clinical Pathologists, March, Dallas, 1978

Difficult Diagnostic Problems in Hemostasis and Thrombosis; Director
American Society of Clinical Pathologists, March, Dallas, 1978

Current Concepts of Hemostasis and Thrombosis, Workshop Director
American Association of Clinical Chemistry, San Francisco, 1978

Modern Concepts and Evaluation of Hemostasis and Thrombosis; Director
American Society of Clinical Pathologists, Fall Meeting, St. Louis, 1978

Disseminated Intravascular Coagulation and Related Syndromes; Director
American Society of Clinical Pathologists, Fall Meeting, St. Louis, 1978

Difficult Diagnostic Problems in Hemostasis and Thrombosis; Director
American Society of Clinical Pathologists, Fall Meeting, St. Louis, 1978

Current Concepts of Hemostasis: Clinical and Laboratory Evaluation & Thrombohemorrhagic Phenomena
American Society of Clinical Pathologist, Regional Cont. Medical Education Program, August, Charleston, South Carolina, 1978

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Recent Concepts and Developments in Evaluating Disorders of Hemostasis and Thrombosis
American Society of Clinical Pathologists, Chicago, 1978

Modern Concepts and Evaluations of Hemostasis and Thrombosis; Director
American Society of Clinical Pathologists, San Diego, 1978

Disseminated Intravascular Coagulation and Related Syndromes; Director
American Society of Clinical Pathologists, San Diego, 1978

Difficult Diagnostic Problems in Hemostasis and Thrombosis; Director
American Society of Clinical Pathologists, San Diego, 1978

Basic Mechanisms of Hemostasis
Grand Rounds (All Staff), Northridge Hospital, Northridge, CA, 1978

Current Status of Mini-heparin
Grand Rounds (All Staff), Northridge Hospital, Northridge, CA, 1978

Hypercoagulability and Thrombosis
Grand Rounds (All Staff), Northridge Hospital, Northridge, CA, 1978

Modern Concepts of Hemostasis and Thrombosis; Director
American Society of Clinical Pathologist (National Seminar), St. Louis, Missouri, 1978

Difficult Diagnostic Problems in Hemostasis and Thrombosis; Director
American Society of Clinical Pathologist, (National Seminar), St. Louis, Missouri, 1978

Antithrombin III: Theory and Clinical Applications
Annual Hemostasis Workshop; John Penner - Director; Ann Arbor, Michigan, 1978

Disseminated Intravascular Coagulation and Related Syndromes; Director
American Society of Clinical Pathologist, St. Louis, Missouri, 1978 (National Seminar)

Course in Hemostasis to Laboratory Technology Students
California State College at Bakersfield, Bakersfield, CA., 1978

Disseminated Intravascular Coagulation and Related Syndromes
Canadian Society of Pathologists, Ontario, Canada, 1978

Current Concepts of Hemostasis and Thrombosis
American Society of Clinical Pathologists (Continuing Education Committee-Seminar) Chicago, Illinois, 1978

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CURRICULUM VITAE: Rodger L. Bick, M.D.

SCIENTIFIC PRESENTATIONS & INVITED SPEAKER/WORKSHOP DIRECTOR (cont.)

Disseminated Intravascular Coagulation and Related Syndromes (Seminar)
California Society of Pathologists, San Francisco, CA., 1978

Current Status of Hemostasis, Thrombosis and Antiplatelet Agents
American Heart Association Annual Meeting, Sacramento, CA., 1979

Difficult Diagnostic Problems in Hemostasis and Thrombosis; Director
American Society of Clinical Pathologist (National Seminar), New Orleans, 1979

Current Concepts of Hemostasis and Thrombosis; Director
American Society of Clinical Pathologist,(National Seminar), New Orleans, 1979

Modern Concepts and Evaluation of Hemostasis and Thrombosis; Director
American Society of Clinical Pathologist, (National Seminar), New Orleans, 1979

Disseminated Intravascular Coagulation and Related Syndromes; Director
American Society of Clinical Pathologist, (National Seminar), New Orleans, 1979

Modern Laboratory Evaluation of Hemostasis and Thrombosis; Director
American Society of Clinical Pathologist, (National Seminar), New Orleans, 1979

Current Concepts in Hemostasis and Thrombosis, Director
California Society of Pathologists, Annual Meeting, San Francisco, CA., 1979

Disseminated Intravascular Coagulation and Related Syndromes
American Society of Clinical Pathologist (Regional Educational Seminar)
Lincoln, Nebraska, 1979

Hypercoagulability
Colorado Association for Continuing Medical Laboratory Education, Colorado, 1979

Current Concepts of Hemostasis and Thrombosis; Director
American Society of Clinical Pathologist (Regional Education Program), Lincoln, Nebraska, 1979

Modern Concepts and Evaluation of Hemostasis and Thrombosis; Director
American Society of Clinical Pathologists, Fall Meeting, Las Vegas, Oct., 1979

Difficult Diagnostic Problems in Hemostasis and Thrombosis; Director
American Society of Clinical Pathologists, Fall Meeting, Las Vegas, Oct., 1979

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SCIENTIFIC PRESENTATIONS & INVITED SPEAKER/WORKSHOP DIRECTOR (cont.)

Disseminated Intravascular Coagulation and Related Syndromes; Director
American Society of Clinical Pathologists, Fall Meeting, Las Vegas, Oct., 1979.

Modern Laboratory Evaluation of Hemostasis and Thrombosis; Director
American Society of Clinical Pathologists, Fall Meeting, Las Vegas, Oct., 1979.

Review Course in Coagulation
American Society of Clinical Pathologists, Winter Meeting, San Diego, CA
January, 1980

Difficult Diagnostic Problems in Hemostasis and Thrombosis, Winter Meeting
American Society of Clinical Pathologists, San Diego, California, January, 1980

Disseminated Intravascular Coagulation
American Society of Clinical Pathologists, Winter Meeting, San Diego, CA
January, 1980

Modern Concepts and Evaluation of Hemostasis and Thrombosis, Spring Meeting
American Society of Clinical pathologists, Atlanta, Georgia, March, 1980

Difficult Diagnostic Problems in Hemostasis and Thrombosis, Spring Meeting
American Society of Clinical Pathologists, Atlanta, Georgia, March, 1980

Disseminated Intravascular Coagulation and Related Syndromes, Spring Meeting
American Society of Clinical Pathologists, Atlanta, Georgia, March, 1980

Clinical Applications of Plasma, Heparin Levels, Antithrombin-III, Plasminogen, and Alpha-2-antiplasmin, San Leandro Doctors Hospital, April 1, 1980

Disseminated Intravascular Coagulation (Grand Rounds Presentation)
Stritch School of Medicine, Loyola University, Maywood, Illinois, May, 1980

Monitoring Heparin Therapy (Surgical Grand Rounds)
Stritch School of Medicine, Loyola University, Maywood, Illinois, May, 1980

Hereditary Hemorrhagic Telangiectasia and Associated Thrombohemorrhagic Defects
Wayne State University, School of Medicine, Detroit, Michigan, May 1980

Disseminated Intravascular Coagulation: A Clinical/Laboratory Correlation in 48 Patients
Wayne State University, School of Medicine, Detroit, Michigan, May 1980

Basic Mechanisms of Hemostasis and Thrombosis
Traveling Consultant in Hematology/Medical Oncology, Tripler Army Medical Center, Honolulu, Hawaii, June, 1980

Hereditary and Acquired Vascular Defects and Hereditary and Acquired Platelet Function Defects
Traveling Consultant in Hemostasis/Medical Oncology, Tripler Army Medical Center, Honolulu, Hawaii, June, 1980

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SCIENTIFIC PRESENTATIONS/INVITED SPEAKER/WORKSHOP DIRECTOR (cont.):

Congenital Coagulation Defects and von Willebrand's Syndrome
Traveling Consultant in Hematology/Medical Oncology, Tripler Army Medical Center, Honolulu, Hawaii, June, 1980.

Disseminated Intravascular Coagulation and Related Syndromes.
Traveling Consultant in Hematology/Medical Oncology, Tripler Army Medical Center, Honolulu, Hawaii, June, 1980.

Hypercoagulability, Anticoagulant Therapy, and Thrombolytic Therapy
Traveling Consultant in Hematology/Medical Oncology, Tripler Army Medical Center, Honolulu, Hawaii, June, 1980.

Current Concepts of Heparin Therapy (Surgical Grand Rounds)
Mercy Hospital, Miami, Florida, September 1980.

Disseminated Intravascular Coagulation (Medical Grand Rounds)
Mercy Hospital, Miami, Florida, September 1980.

Hemostasis and Bleeding Disorders in Problem Patients (Grand Rounds)
Mt. Sinai Medical Center, Miami Beach, FL September 1980.

Clinical Applications of Measuring Antithrombin III Levels; With Emphasis on its Relationship to Monitoring Heparin Therapy. (Florida Soc. Pathologists)
Mt. Sinai Medical Center, Miami Beach, Fl September 1980.

Current Concepts of Heparin Therapy
Valley Medical Center, Fresno, CA September 1980.

Disseminated Intravascular Coagulation
Valley Medical Center, Fresno, CA. September 1980.

Disseminated Intravascular Coagulation and Related Syndromes
American Society of Clinical Pathologists, Fall Meeting
St. Louis, October, 1980.

Difficult Diagnostic Problems in Hemostasis and Thrombosis
American Society of Clinical Pathologists, Fall Meeting
St. Louis October 1980.

Modern Concepts and Evaluation of Hemostasis and Thrombosis
American Society of Clinical Pathologists, Fall Meeting
St. Louis October 1980.

Monitoring Heparin Therapy & Mini-Heparin Therapy (Grand Rounds)
Dameron Hospital Association, Stockton, CA February 1981.

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SCIENTIFIC PRESENTATIONS/INVITED SPEAKER/WORKSHOP DIRECTOR (cont.):

Syndromes of DIC
American Dade Rocky Mountain Hemostasis Conference
Denver, Colorado March 1981.

Current Controversies About Heparin Therapy
American Dade Rocky Mountain Hemostasis Conference
Denver, Colorado March 1981.

Modern Concepts and Evaluation of Hemostasis and Thrombosis
American Society of Clinical Pathologists, Spring Meeting
San Diego, CA March 1981.

Difficult Diagnostic Problems in Hemostasis and Thrombosis
American Society of Clinical Pathologists, Spring Meeting
San Diego, CA March 1981.

The Need for Tests of Hypercoagulability
American Dade Hypercoagulability Seminar
Miami, FL March 1981.

Disseminated Intravascular Coagulation and Related Syndromes
Washington State Society for Medical Technology, Spring Seminar
Spokane, Washington April 1981.

Antithrombin-III
California Blood Bank System Annual Meeting
Los Angeles, CA April 1981.

Antiplatelet and Mini-Heparin Therapy
Methodist Hospital of Southern California
Arcadia, CA May 1981.

Hemostasis, Coagulation, DIC and Proteolysis
American Society of Clinical Pathologists, Regional Educational Program
Los Angeles, CA June 1981.

Acquired AT-III Deficiencies that Occur with Surgery; Surgical Grand Rounds
The Medical College of Wisconsin, Milwaukee, Wisconsin, June 1981.

AT-III and DIC: Hematology Conference Rounds
The Medical College of Wisconsin, Milwaukee, Wisconsin, June 1981.

Antithrombin-III/Heparin Interrelationships and Methods of Testing for AT-III
Pathology Monthly Concerence, Milwaukee, Wisconsin, June 1981.

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SCIENTIFIC PRESENTATIONS/INVITED SPEAKER/WORKSHOP DIRECTOR (cont.)

Hereditary Hemorrhagic Telangiectasia and Disseminated Intravascular Coagulation: A New Clinical Syndrome.
VIIIth International Congress on Thrombosis and Haemostasis
Toronto, Ontario, Canada July 1981.

Piracetam: A New Platelet Suppressing Drug.
VIIIth International Congress on Thrombosis and Haemostasis
Toronto, Ontario, Canada July 1981.

The Effect of Piracetam in Preventing Recurrent Deep Venous Thrombosis.
VIIIth International Congress on Thrombosis and Haemostasis
Toronto, Ontario, Canada, July 1981.

A Comparison of Chromogenic, Fluorogenic, and Fibrinogen Substrate Assays for the Determination of Plasma Heparin
VIIIth International Congress on Thrombosis and Haemostasis
Toronto, Ontario, Canada, July 1981.

Studies Related to Tobacco Glycoprotein: A Claimed Activator of Coagulation, Fibrinolysis, Complement, Kinin, and a Claimed Allergen.
VIIIth International Congress on Thrombosis and Haemostasis
Toronto, Ontario, Canada, July 1981.

Clinicians and New Methods in Coagulation Testing
Annual Meeting of the American Association of Clinical Chemists (Invited Speaker).
St. Louis, Missouri July 1981.

A Review of Hemostasis and Thrombosis
The Colorado Association for Continuing Medical Laboratory Education
Dillon, Colorado July 1981.

Hemostasis, Coagulation, Disseminated Intravascular Coagulation and Proteolysis
American Society of Clinical Pathologists
Atlanta, Georgia September 1981

Hemostasis, Coagulation, Disseminated Intravascular Coagulation and Proteolysis
American Society of Clinical Pathologists
Silver Springs, Maryland, September 1981

Clinical Applications of AT-III
Surbaban Hospital, Silver Springs, Maryland Grand Rounds
September 1981

DIC and Bleeding Problems Associated with Open Heart Surgery
National Naval Medical Center Grand Rounds
Bethesda, Maryland September 1981

SCIENTIFIC PRESENTATIONS/INVITED SPEAKER/WORKSHOP DIRECTOR (cont.):

Modern Concepts and Evaluations of Hemostasis and Thrombosis
American Society of Clinical Pathologists and College of American Pathologists
Fall Meeting, Las Vegas, Nevada, October, 1981

Difficult Diagnostic Problems in Hemostasis and Thrombosis
American Society of Clinical Pathologists and College of American Pathologists
Fall Meeting, Las Vegas, Nevada, October, 1981

New Methods in Coagulation Testing - Scientific Exhibit
American Society of Clinical Pathologists and College of American Pathologists
Fall Meeting, Las Vegas, Nevada, October 1981

Review Course in Coagulation
American Society of Clinical Pathologists, CAAMA-CCE Joint ASCP Project
Los Angeles, CA November, 1981

MEMBERSHIPS:

International Society of Haemotology

International Society on Thrombosis and Haemostasis

Thrombosis Council-American Heart Association

American College of Angiology (Fellow)

International College of Angiology (Fellow)

American Association for the Study of Neoplastic Disease.

American Association for Clinical Research

Central Oncology Group (Clinical Investigator) 1974-1975

American Society of Mammologists

Nigerian Haematology Society

Southwest Oncology Group (Clinical Investigator) 1976-Nov. 1977.

California College Honor Society

American Cancer Society

International Association for the Study of Lung Cancer (Founding Member)

Federation of American Societists

American Geriatrics Society (Founding Fellow)

Professional Education Committee, American Cancer Society, Los Angeles Unit

Research and Grant Peer Review Committee, American Heart Association

Consultant: American Tobacco Research Council

Chairman: Professional Education Committee, American Heart Association, Kern County Unit

Member: Professional Education Committee: American Cancer Society, Kern County Unit

MEMBERSHIPS (cont.)

American Association for the Advancement of Science

New York Academy of Sciences

American College of Physicians

American Society of Clinical Pathologists

American Society of Hematology

American Heart Association (Council on Thrombosis, Circulation, and Atherosclerosis)

Association of Clinical Scientists

California Thoracic Society

Who's Who in the West

Am Phys Soc; Am Asn Physics Teachers; [illegible] Soc Physics & Neutron [illegible]. Res: Interaction of radiation with matter; neutron physics; use of radiation in medicine and biology; cancer therapy with fast neutron beams; neutron radiation dosimetry. Mailing Add: Dept of Radiol Univ Hosp Seattle WA 98195

BICHTELER, KLAUS RICHARD, b Leipzig, Ger, Mar 15, 38; m 68; c 1. MATHEMATICS. Educ: Univ Hamburg, dipl, 63, PhD(gen relativity), 65. Prof Exp: Asst math, Univ Heidelberg, 65-68; asst prof, Southwest Ctr Advan Studies, 66-69; asst prof, 69-71, ASSOC PROF MATH, UNIV TEX, AUSTIN, 71- Mem: Am Math Soc. Res: General relativity; representation theory of topological groups and algebras. Mailing Add: Dept of Math Univ of Tex Austin TX 78712

BICK, GEORGE HERMAN, b Negreet, La, Sept 23, 14; m 43; c 2. ENTOMOLOGY, ZOOLOGY. Educ: Tulane Univ, BS, 36, MS, 38; Cornell Univ, PhD(entom), 47. Prof Exp: Inspector, Food & Drug Admin, USDA, La, 38-41; field biologist, State Dept Conserv, La, 41-42; asst prof, Clark Miss, 47-48 & Tulane Univ, 48-56; assoc prof, Univ Southwestern La, 56-59; prof, Clarion State Col, 59-60; PROF BIOL, ST MARY'S COL, IND, 60- Mem: Fel AAAS; Entom Soc Am. Res: Biology of Odonata. Mailing Add: Dept of Biol St Mary's Col Notre Dame IN 46556

BICK, KATHERINE LIVINGSTONE, b PEI, Can; US citizen; c 2. NEUROBIOLOGY, HISTOCHEMISTRY. Educ: Acadia Univ, BSc, 51, MSc, 52; Brown Univ, PhD(biol), 57. Prof Exp: Res assoc biochem, Colby Med Res Lab, Univ Western Ont, 52-54; asst res [illegible], Sch Med, Univ Calif, Los Angeles, 59-61; asst prof biol, Calif State Univ, Northridge, 61-68 & Georgetown Univ, 70-76; ASST DIR NEUROL DIS PROG, NAT INST NEUROL & COMMUN DIS & STROKE, 76- Mem: Am Acad Neurol; AAAS; Am Soc Zool. Res: Alzheimer's disease, senile dementia and related degenerative disorders of the nervous system; demyelinating disorders; histochemistry of [illegible]; histochemistry of calcification. Mailing Add: Rm 716 Fed Bldg Nat Inst Neurol & Commun Dis & Stroke 7550 Wisconsin Ave Bethesda MD 20014

BICK, KENNETH F, b Janesville, Wis, Feb 14, 32; m 58; c 3. GEOLOGY. Educ: Yale Univ, BS, 54, MS, 56, PhD(geol), 58. Prof Exp: Asst prof geol, Washington & Lee Univ, 58-61; assoc prof, 61-66, chmn dept geol, 62-66; PROF GEOL, COL WILLIAM & MARY, 66- Mem: AAAS; Geol Soc Am; Am Asn Petrol Geologists; Am Geophys Union. Res: Structural and stratigraphic geology; application of computers to geological problems. Mailing Add: Dept of Geol Col of William & Mary Williamsburg VA 23185

BICK, MICHAEL DAVID, b Stockton, Calif, Jan 6, 45; m 66; c 2. MOLECULAR BIOLOGY, HEMATOLOGY. Educ: Univ Puget Sound, BA, 66; Univ Southern Calif, PhD(cell & molecular biol), 71. Prof Exp: Fel biochem, Harvard Med Sch, 71-72, fel biol chem, 72-74, fel clin genet, Harvard Med Sch & Children's Hosp Med Ctr, 74; asst mem, Roche Inst Molecular Biol, 74-78; SCI DIR, SAN JOAQUIN HEMAT & ONCOL LAB, INC, 78-, DIR QUAL CONTROL, PAC HEMOSTASIS LAB, INC, 78- Concurrent Pos: Fel, Damon Runyon Cancer Found, 71-73 & Bureau Med Found, Mass, 73-74; consult, Bay Area Hemat & Oncol Labs, Inc, 77- Mem: Am Soc Microbiol; Am Soc Cell Biol. Res: Regulation of gene expression in cultured cell lines; biochemistry of plasma proteins; development of clinical assays in hematology and oncology. Mailing Add: San Joaquin Hemat & Oncol Lab Inc 2727 Eye St Bakersfield CA 93301

BICK, PETER HAMILTON, b Toledo, Ohio, Sept 30, 48; m 71; c 1. IMMUNOLOGY. Educ: Albion Col, AB, 70; Univ Mich, PhD(microbiol), 75. Prof Exp: Guest scientist immunol, Div Immunobiol, Karolinska Inst, Stockholm, Sweden, 75-77; fel genetics, Wash Univ, 77-78; ASST PROF IMMUNOL, DEPT MICROBIOL, MED COL VA, 78- Concurrent Pos: Arthritis Found fel, 75-78. Res: Activation and regulation of immunocompetent cells. Mailing Add: Dept of Microbiol Box 647 MCV Sta Med Col of Va Richmond VA 23298

BICK, RODGER LEE, b San Francisco, Calif, May 21, 42. HEMATOLOGY, ONCOLOGY. Educ: Univ Calif, Irvine, MD, 70. Prof Exp: From intern to resident internal med, Kern Gen Hosp, Bakersfield, Calif, 70-72, fel, 72-73; MEM FAC HEME-ONCOL, UNIV CALIF CTR HEALTH SCI LOS ANGELES, 73-, MED DIR, BAY AREA HEMAT ONCOL RES LABS, 75- Concurrent Pos: Dir med educ & hemat-oncol, Kern Gen Hosp, 73-74; attend hematologist, Santa Monica Med Ctr, Calif, 74-, dir oncol, 75-; attend hematologist, St John's Health Sci Ctr, Santa Monica & Univ Calif Ctr Health Sci, Los Angeles, 74-; mem thrombosis coun, Am Heart Asn. Mem: [illegible] Soc Thrombosis & Hemostasis; Am Soc Hemat; Am Cancer Soc. Res: Hemostasis and blood coagulation; alterations of hemostasis associated with cardiopulmonary bypass; hypercoagulability and thrombosis; acquired antithrombin-III deficiency; alterations of hemostasis associated with malignancy; nature of disseminated intravascular coagulation. Mailing Add: San Joaquin Hemat Oncol Group 2727 Eye St Bakersfield CA 93301

BICK, THEODORE A, b Brooklyn, NY, Dec 18, 38; m 56; c 4. MATHEMATICS. Educ: Union Col, BS, 58; Univ Rochester, MS, 60, PhD(ergodic theory), 64. Prof Exp: Instr math, Hobart & William Smith Cols, 61-64; from asst prof to assoc prof, 64-66, ASSOC PROF MATH, UNION COL, NY, 66- Mem: Am Math Soc; Math Asn Am. Res: Ergodic theory; functional analysis; real variable theory; measure and integration. Mailing Add: Dept of Math Union Col Schenectady NY 12308

BICKART, THEODORE ALBERT, b Flushing, NY, Aug 25, 35; m 58; c 3. ELECTRICAL ENGINEERING. Educ: Johns Hopkins Univ, BES, 57, MSE, 58, DEng, 60. Prof Exp: Instr elec eng, Johns Hopkins Univ, 58-59, lectr, 59-61; from asst prof to assoc prof, 63-70, PROF ELEC ENG, SYRACUSE UNIV, 70- Concurrent Pos: Vis scholar, dept elec eng & comput sci, Univ Calif, Berkeley, 77. Mem: Fel Inst Elec & Electronics Engrs; Am Math Soc; Soc Indust & Appl Math; Asn Comput Mach. Res: Nonlinear systems analysis; linear systems analysis; system simulation; numerical analysis & functional analysis in systems analysis; stability theory. Mailing Add: Dept of Elec & Comput Eng Link Hall Syracuse Univ Syracuse NY 13210

BICKEL, EDWIN DAVID, b Louisville, Ky, Nov 11, 41; m 67; c 2. ECONOMIC GEOLOGY, ENVIRONMENTAL GEOLOGY. Educ: Univ Louisville, AB, 63, MSc, 65; Ohio State Univ, PhD(geol), 70. Prof Exp: Asst prof geol, 70-73, dir comp educ, Minot State Col, 73-76; ADMINR GEOL, N AM COAL CORP, 77- Concurrent Pos: Work-group researcher, Northern Great Plains Resources Prog, 74-75; contracted malacologist, Off Endangered Species, US Fish & Wildlife Serv, 74-; consult, 75-77; prin investr, NDak Regional Environ Assessment Prog, 76-77. Mem: Am Asn Petrol Geologists; Am Malacol Union; Paleont Soc; Sigma Xi; AAAS. Res: Malacology, systematics and ecology of living and fossil non-marine Mollusca; Cretaceous-Tertiary biostratigraphy; coal stratigraphy; socio-economic and environmental impacts of energy development. Mailing Add: N Am Coal Corp Kirkwood Off Tower Bismarck ND 58501

BICKEL, PETER J, b Bucharest, Romania, Sept 21, 40; US citizen; m 64; c 2. MATHEMATICAL STATISTICS. Educ: Univ Calif, Berkeley, AB, 60, MA, 61, PhD(statist), 63. Prof Exp: From asst prof to assoc prof statist, 63-70, chmn dept, 73-76, PROF STATIST, UNIV CALIF, BERKELEY, 70- Concurrent Pos: Lectr, Imp Col, Univ London, 65-66; J S Guggenheim Found fel, 70; NATO sr sci fel, 74-75. Mem: Fel Inst Math Statist; Am Statist Asn; Royal Statist Soc. Res: Nonparametric statistics; robustness; asymptotic methods. Mailing Add: Dept of Statist Univ of Calif Berkeley CA 94720

BICKEL, ROBERT JOHN, b Louisville, Ky, Nov 8, 16; m 42. MATHEMATICS. Educ: Univ Louisville, AB, 37; Northwestern Univ, MA, 41; Univ Pittsburgh, PhD(math), [illegible] Prof Exp: Teacher, pub schs, Ky, 37-41; from instr to assoc prof, 46-66, [illegible] 66-69, 73-74, PROF MATH, DREXEL UNIV, 66-, ASSOC HEAD DEPT, 69- Mem: Math Asn Am; Soc Indust & Appl Math (treas, 55-62). Res: Analysis; [illegible] divergent series. Mailing Add: Dept of Math Drexel Univ Philadelphia PA

BICKEL, THOMAS FULCHER, b Detroit, Mich, Nov 20, 37; m 64. ALGEBRA. Educ: Univ Mich, BS, 59, MA, 60, PhD(math), 65. Prof Exp: Instr math, [illegible] 65-67, asst prof, 67-73, ASSOC PROF MATH, DARTMOUTH COL, 73- Mem: [illegible] Soc. Res: Finite group theory; theory of finite groups of Lie type; [illegible] Mailing Add: Dept of Math Dartmouth Col Hanover NH 03755

BICKEL, WILLIAM SAMUEL, b Osceola, Pa, June 8, 37. EXPERIMENTAL PHYSICS, SPECTROSCOPY. Educ: Pa State Univ, BS, 59, PhD(physics), 65. Prof Exp: [illegible] physics, 65; from asst prof to assoc prof, 65-75, PROF PHYSICS, UNIV ARIZ, 75- Mem: Am Phys Soc. Res: Spectroscopic diagnostics; atomic physics; spectroscopy of [illegible] ions; measurement of atomic lives of excited ions; vacuum ultraviolet and [illegible] spectroscopy; biophysics; time-resolved polarized light scattering from [illegible] macromolecules. Mailing Add: Dept of Physics Univ of Ariz Tucson AZ

BICKELHAUPT, R(OY) E(DWARD), b Waterloo, Ill, Jan 14, 28; m 48; c 1. CERAMIC ENGINEERING. Educ: Univ Ill, BS, 50, PhD(ceramic eng), 63. Prof Exp: Instr ceramic eng, Clemson Col, 50-52; engr, Prod Div, Bendix Corp, [illegible] eng chief engr ceramic eng, Marshall Eclipse Div, 55-59; res assoc ceramic eng, Ill, 59-63; SR CERAMIC ENGR, SOUTHERN RES INST, 63- Mem: Am Soc [illegible]; Am Ceramic Soc. Res: Mechanical friction; thermophysical properties of [illegible] composites; chemical diffusion in oxide systems; fracture mechanisms of [illegible]; electrostatic precipitation; electrical resistivity of particulates. Mailing Add: Southern Res Inst 2000 Ninth Ave South Birmingham AL 35205

BICKERMAN, HYLAN A, b New York, NY, Oct 26, 13; m 41; c 2. RESPIRATORY PHYSIOLOGY, CLINICAL PHARMACOLOGY. Educ: Columbia Univ, BA, 35; NY Univ, MD, 39. Prof Exp: CHIEF ASTHMA-EMPHYSEMA CLIN, COLUMBIA-PRESBY MED CTR, 61- Concurrent Pos: Res fel med & chest [illegible], Columbia Univ Res Serv, Goldwater Mem Hosp, New York, 42-47; vis physician, [illegible]; chief med serv, Cushing Gen Hosp, Boston, 45-46; asst clin prof, Col Physicians & Surgeons, Columbia Univ, 57-58, assoc clin prof, 58-73, clin prof, 73-; consult, St [illegible] Hosp, New York, 65-71, Brookhaven Mem Hosp, Suffolk County, NY & Arden Hill [illegible], Monroe, NY, 68-; dir respiratory lab & inhalation ther dept, Francis Delafield Hosp, New York, 68-75; panelist on emphysema, Int Cong Chest Dis, Lausanne, Switz, [illegible] physician, Presby Hosp, New York, 70-78, attend physician, 78-; [illegible] Drug Admin, 72-76; dir pulmonary function labs, Doctors Hosp, New York. Honors & Awards: Golden Tree of Life Award, Am Asn Inhalation Therapists, 67. Mem: AAAS; AMA; fel Am Col Chest Physicians; fel Am Col Physicians; Am Thoracic Soc. Res: Respiratory physiology and management of patients with chronic obstructive lung disease, especially pharmacologic therapy; development of various modalities of inhalation therapy including aerosols and pressure breathing. Mailing Add: Col of Physicians & Surgeons Columbia Univ New York NY 10027

BICKERSTAFF, THOMAS ALTON, mathematics, see 12th edition

BICKERT, WILLIAM GEORGE, b Bismarck, NDak, Apr 9, 37; m 60; c 2. AGRICULTURAL ENGINEERING. Educ: NDak State Univ, BS, 59, MS, [illegible] State Univ, PhD(agr eng), 64. Prof Exp: Res asst agr eng, NDak State Univ, 59-60; [illegible] res asst & instr to assoc prof, 61-72, PROF AGR ENG, MICH STATE UNIV, 72- Mem: Am Soc Agr Engrs. Res: Milking systems, equipment, design, labor utilization, [illegible] production facilities, ventilation, structures, systems layout, environmental [illegible] Mailing Add: Dept of Agr Eng Mich State Univ East Lansing MI 48824

BICKERTON, ROBERT KEITH, b East Liverpool, Ohio, Oct 1, 30; m 59; c [illegible] PHARMACOLOGY. Educ: Univ Pittsburgh, BS, 56, MS, 58, PhD(pharmacol), [illegible] Prof Exp: Sr res pharmacologist, 60-62, unit leader, 62-63, from asst chief to chief [illegible] pharmacol, 63-65, dir pharmacodynamics div, 65-68, sr res, 68-69, vpres res, 69-72, [illegible] sci affairs, 72-77, SR V PRES SCI AFFAIRS, NORWICH PHARMACAL CO, 77- Mem: Am Pharmaceut Asn; Am Soc Pharmacol & Exp Therapeut. Res: Mechanism of [illegible] development of antihypertensive drugs. Mailing Add: Norwich-Eaton Pharmaceut [illegible] 191 Norwich NY 13815

BICKFORD, LAWRENCE RICHARDSON, b Elmira, NY, Nov 24, 21; m 43; c 3. SOLID STATE PHYSICS. Educ: Alfred Univ, BS, 43; Mass Inst Technol, PhD(physics), 49. Prof Exp: Assoc prof physics, State Univ NY Col Ceramics, Alfred, 49-54; sr physicist, [illegible] 54-63, dir gen sci, Res Ctr, 62-63, dir res lab, Tokyo, Japan, 63-65; dir memory & [illegible] Res Lab, NY, 65-70; mgr mat sci & technol, 70-73, res staff mem, IBM Res Lab, [illegible] 73-77, PROG DIR TECH COMMUN, IBM WHITE PLAINS, NY, 77- Mem: Fel Am Phys Soc; Inst Elec & Electronics Engrs. Res: Ferrites; titanates; glass; organic [illegible]; contact effects on conductivity in anisotropic organic conductors; [illegible] Mailing Add: IBM 10 705 44 Broadway White Plains NY 10601

BICKFORD, MARION EUGENE, b Memphis, Tenn, Aug 30, 32; m 56; c [illegible] PETROLOGY, GEOCHEMISTRY. Educ: Cornell Col, BA, 54; Univ Ill, MS, [illegible] PhD(geol), 60. Prof Exp: Asst prof geol, San Fernando Valley State Col, 60-62; asst geophysicist, Inst Geophys, Univ Calif, Los Angeles, 62-64; from assoc prof to prof geol, 64-73, PROF GEOL, UNIV KANS, 73- Mem: Mineral Soc Am; Geol Soc Am; Am Geophys Union; Geochem Soc. Res: Geochronology and petrology of the Precambrian [illegible] southwestern Missouri; isotopic and elemental studies of magma genesis. Mailing Add: Dept of Geol Univ of Kans Lawrence KS 66045

BICKFORD, REGINALD G, b Bexwood, Eng, Jan 20, 13; nat US; m 42; c [illegible] NEUROSCIENCES. Educ: Cambridge Univ, BA, MD & BCh, 36; FRCP, 71. Prof Exp: Mayo res assoc, Univ Minn, 46-48, from assoc prof to prof physiol, 53-69; PROF NEUROSCI, SCH MED & HEAD EEG LAB, UNIV HOSP, UNIV CALIF, SAN DIEGO, 69- Concurrent Pos: Med Res Coun fel, Univ London, 37-40; consult EEG, [illegible] Clin, 46-69; mem neurol study sect, USPHS, 60-64, mem comput study sect, [illegible] adv coun epilepsy, 64-70; mem clin pract coun drug in Soviet Union, 68 & mem [illegible] prog adv comt, Nat Inst Neurol Dis & Stroke, 73- Mem: Am EEG Soc (pres, [illegible]); Physiol Soc; Int League Against Epilepsy; Am Neurol Asn. Res: Electrical activity of brain in man and animals; experimentally produced changes and their relation to [illegible] and psychological effects; computer analysis and automation of the [illegible] Mailing Add: Dept of Neurosci Univ of Calif Sch of Med La Jolla CA

BICKING, JOHN BERTE, b Reading, Pa, Feb 11, 20; m 49; c 2. ORGANIC CHEMISTRY. Educ: Drexel Inst, BS, 42; Univ Del, MS, 48, PhD(chem), 53. Prof Exp: Control chemist, Gen Chem Div, Allied Chem & Dye Corp, 46-47; SR INVESTR, MERCK SHARP & DOHME RES LABS DIV, MERCK & CO, INC, 48- Mem: Am Chem Soc. Res: Synthetic organic chemistry. Mailing Add: 622 Salford Ave Lansdale PA 19446

BICKLEY, HARMON C, b Detroit, Mich, May 1, 30; m 56, 68; c 4. PATHOLOGY, DENTISTRY. Educ: Univ Mich, DDS, 59; Univ Rochester, PhD(exp path), 64. Pro

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AMERICAN MEN & WOMEN OF SCIENCE

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STATEMENT OF THEODORE H. BLAU, PH.D.
PRESENTED BEFORE
SUBCOMMITTEE ON HEALTH AND THE ENVIRONMENT
HOUSE OF REPRESENTATIVES

Thank you Mr. Chairman for the opportunity of appearing before your Subcommittee. My name is Theodore H. Blau. I hold the Doctorate in Psychology, received from the Pennsylvania State University in 1951. I have been in independent practice of clinical psychology and consulting psychology in Tampa, Floria, since 1953. Formerly, I was Professor of Psychiatry at the Medical School of the University of South Florida. I hold a Diplomate in clinical psychology from the American Board of Professional Psychology as well as the Diploma from the American Board of Forensic Psychology.

I am a member and past President of the American Psychological Association and am currently President of the American Psychological Foundation. I am also a member of the Evaluation Research Society and have conducted evaluation studies for private corporations as well as evaluation research for various branches of the U.S. military and the government. I am the author of one book, a number of chapters and approximately fifty articles.

For several years, I have been involved in an evaluation of the research and current state of knowledge about the psychological aspects of cigarette smoking. This evaluation includes a

THOROUGH REVIEW OF HISTORICAL FACTORS, CURRENT THEORIES AND RESEARCH, QUALITY OF THE RESEARCH AND PUBLICATIONS PERTAINING TO DEPENDENCE, ADDICTION, LABORATORY AND APPLIED RESEARCH AND CULTURAL ASPECTS OF SMOKING BEHAVIOR. IN ADDITION TO THIS LITERATURE REVIEW, I HAVE ALSO COMMUNICATED WITH ACTIVE RESEARCHERS WORKING IN THE AREA. I AM PRESENTLY WRITING THE SECTION ENTITLED "SMOKING BEHAVIOR" THAT WILL BE PUBLISHED IN THE *WILEY ENCYCLOPEDIA OF PSYCHOLOGY*.

IT IS MY UNDERSTANDING THAT PROPOSED BILL HR 5653 WOULD MANDATE A WARNING ON CIGARETTE PACKAGES STATING THAT CIGARETTE SMOKING IS ADDICTIVE. I WILL ADDRESS MYSELF TODAY TO THAT CONCLUSION.

FOR ALMOST 400 YEARS THE SMOKING HABIT HAS BEEN AN ISSUE NEVER LACKING PROPONENTS AS WELL AS OPPONENTS. SOME WHO SMOKE SEEM UNABLE TO GIVE UP THE CIGARETTE HABIT AT LEAST DURING CERTAIN STAGES OF THEIR LIVES. OTHERS CAN QUIT WITH APPARENT EASE. FEW SPECIFIC AREAS OF APPLIED PSYCHOLOGICAL RESEARCH HAVE RECEIVED GREATER ATTENTION THAN THE CIGARETTE SMOKING HABIT. DESPITE THIS INTENSE SCIENTIFIC FOCUS ON THE BEHAVIORAL AS WELL AS PHYSIOLOGICAL ASPECTS OF CIGARETTE SMOKING, NO GENERAL AGREEMENT AMONG SCIENTISTS EXISTS AS TO THE QUESTION OF WHETHER CIGARETTES OR THEIR CONTENTS ARE ADDICTIVE. THE BEHAVIOR AND RESPONSES OF CIGARETTE SMOKERS ARE QUITE DIFFERENT THAN THOSE OBSERVED IN INDIVIDUALS WHO ARE

ADDICTED TO HEROIN AND OTHER SUBSTANCES THAT ARE DEMONSTRABLY ADDICTIVE.

THE SCIENTIFIC LITERATURE DEMONSTRATES THE FOLLOWING:

THERE IS APPARENTLY NO DIFFERENCE IN THE SEVERITY OF CESSATION RESPONSES BETWEEN LIGHT SMOKERS AND HEAVY SMOKERS. RESEARCH ALSO HAS SHOWN THAT THESE EFFECTS ARE FELT MORE SEVERELY BY THOSE WHO REDUCED BUT DID NOT STOP COMPLETELY THAN BY THOSE WHO ABSTAINED TOTALLY. A THIRD ASPECT REPORTED BY VARIOUS RESEARCHERS IS THAT SMOKING CESSATION EFFECTS ARE RELATIVELY MILD AND IN SOME CASES NON-EXISTENT. THEY ARE OF A NATURE WHICH MIGHT BE EXPECTED TO FOLLOW THE LOSS OF PSYCHOLOGICAL REWARDS FROM ANY LOVED OBJECT TO WHICH A PERSON WAS LONG ACCUSTOMED AND WHICH COULD BE CONCEIVED OF AS FORMING A PART OF THE INDIVIDUAL SELF-IMAGE.

IT HAS BEEN REPORTED THAT MANY SMOKERS ARE ABLE TO REFRAIN FROM SMOKING FOR RELATIVELY LONG PERIODS OF TIME FOR PRACTICAL, SAFETY OR RELIGIOUS REASONS AND TO DO SO WITHOUT APPARENT DISCOMFORT. SOME EXAMPLES ARE COAL MINERS WHO MAY NOT SMOKE AT THE PITFACE, ORTHODOX JEWS WHO GIVE UP SMOKING AT SUNDOWN ON FRIDAY AND CEASE SMOKING UNTIL SUNDOWN ON SATURDAY, AND SO FORTH. SUCH BEHAVIOR DOES NOT FIT CONVENTIONAL VIEWS OF ADDICTION.

WHEREAS THE EFFECTS OF USE AND WITHDRAWAL ARE CONSISTENT AND PREDICTABLE WITH KNOWN ADDICTIVE SUBSTANCES DESPITE A WIDE

RANGE OF USES, DESCRIPTIONS OF TOBACCO EFFECTS ARE EXTREMELY VARIED AND INCONSISTENT. IN THIS REGARD, TOBACCO USE IS MORE LIKE THE USE OF CAFFEINE RATHER THAN ALCOHOL OR OPIATES. IT HAS BEEN REPORTED (COSTA, 1980) THAT THE CONTINUANCE OF SMOKING APPEARS MORE RELATED TO A WIDE RANGE OF PSYCHOSOCIAL MOTIVES SUCH AS PLEASURE, STIMULATION, SENSORY MOTOR MANIPULATION, AND REDUCTION OF NEGATIVE AFFECT THAN AN ADDICTION FACTOR.

THE UNITED STATES DEPARTMENT OF H.E.W. REPORTED IN 1977 THAT 95 PERCENT OF THOSE PEOPLE WHO QUIT SMOKING DO SO ON THEIR OWN. THIS IS NOT ONLY A PHENOMENA IN SHARP CONTRAST TO EXPERIENCE WITH DEMONSTRABLY ADDICTIVE SUBSTANCES BUT ONE WHICH REMAINS TO BE EXPLORED.

CLEARLY, MANY AREAS OF SMOKING BEHAVIOR ARE AS YET UNEXPLORED INCLUDING WHY SOME SMOKE VERY HEAVILY AND SOME DO NOT. CAREFULLY CONTROLLED ANIMAL STUDIES MUST BE DONE BEFORE SCIENTIFIC DESCRIPTION OF THE SMOKING HABIT CAN BE EXPECTED. PROMISING WORK WITH RHESUS MONKEYS HAS BEEN REPORTED (KIYOSHI AND YANAGITA) IN JAPAN AND RESEARCHERS AT THE SOUTHWEST RESEARCH INSTITUTE (ROGERS) HAVE REPORTED CONDITIONING LABORATORY BABOONS TO SMOKE CIGARETTES. THESE DEVELOPMENTS SUGGEST THAT WE MAY BE ABLE TO STUDY CIGARETTE SMOKING IN A PROPERLY CONTROLLED LABORATORY SETTING. UNTIL SUCH CONTROLLED ANIMAL EXPERIMENTS WITH RELIABLE MODELS OF SELF-ADMINISTRATION OF TOBACCO CAN BE DEVISED AND CONDUCTED AS THEY

HAVE BEEN WITH MORPHINE, COCAINE AND ALCOHOL, THE CONCEPT OF TOBACCO ADDICTION WILL PROBABLY REMAIN A HYPOTHETICAL CONCEPT ONLY PARTLY UNDERSTOOD (JARVIK, 1977). AND SO, GENTLEMEN, AT THIS TIME THE SCIENTIFIC DATA DO NOT SUPPORT THE STATEMENT: CIGARETTE SMOKING IS ADDICTIVE.

I WOULD NOW LIKE TO DISCUSS VERY BRIEFLY MY CLINICAL EXPERIENCE WITH SMOKING. DURING THE PAST 30 YEARS, I HAVE WORKED AND CONTINUE TO WORK WITH PEOPLE WHO SMOKE CIGARETTES. IN REVIEWING MY EXPERIENCE WITH THE THOUSANDS OF PEOPLE THAT I HAVE SEEN PROFESSIONALLY, MANY OF WHOM ARE HEAVY SMOKERS, SOME CONTINUED, SOME QUIT WITH STRESS, AND SOME QUIT WITH LITTLE OR NO STRESS. THESE PATIENTS IN NO WAY ACTED LIKE THE PATIENTS THAT I HAVE SEEN WHO STRUGGLE TO BE RELEASED FROM THE ADDICTIONS OF OPIATES OR ALCOHOL.

CIGARETTE SMOKERS ARE VERY ATTACHED TO THEIR SMOKING BEHAVIOR. THEY ARE OFTEN ANNOYED OR DISTRESSED WHEN THEY ARE NOT ALLOWED TO SMOKE. HOWEVER, I HAVE NOTED EQUALLY STRONG ATTACHMENTS TO TENNIS, JOGGING, CANDY, ROCK MUSIC, COCA-COLA, MEMBERS OF THE OPPOSITE SEX, AND HAMBURGERS IN MY ADOLESCENT AND ADULT PATIENTS. WITH CHILDREN, ONE SEES VERY STRONG ATTACHMENTS TO PLAYMATES, PARENTS, CERTAIN ARTICLES OF CLOTHING, TV, BLANKETS AND TEDDY BEARS. REMOVAL FROM THESE ACTIVITIES, PERSONS OR OBJECTS CAN RESULT IN AGITATION, SLEEPLESSNESS, IRRITATION, DEPRESSION AND OTHER UNCOMFORTABLE SYMPTOMS. THEY VARY CONSIDERABLY IN INTENSITY

AND DURATION, AS DO EFFECTS FOLLOWING ABSTINENCE FROM TOBACCO SMOKING.

I, MYSELF, SMOKED CIGARETTES FOR 24 YEARS. TWO TO THREE PACKAGES A DAY FOR THE LAST TEN YEARS OF THE HABIT. I STOPPED AT THE AGE OF 35 AND HAVE NOT SMOKED SINCE. I WAS SOMEWHAT UNCOMFORTABLE FOR A SHORT PERIOD OF TIME, BUT WAS NEVER HIGHLY UNCOMFORTABLE. THE CRAVING NEVER REACHED THE LEVEL I HAVE EXPERIENCED OR OBSERVED IN MEDICAL WEIGHT LOSS PROGRAMS. IN SHORT, ALTHOUGH CIGARETTE SMOKING IS A COMMON AND PERVASIVE HABIT, I CAN FIND NO CONVINCING BASIS IN THE SCIENTIFIC LITERATURE OR IN MY OWN PROFESSIONAL CLINICAL EXPERIENCE TO JUSTIFY LABELING IT OR TREATING IT AS AN ADDICTION.

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[illegible]
Grant Proposals
Statistical Analyses
Medico-legal Assistance

STATEMENT OF WALTER M. BOOKER, PH.D.

My name is Walter M. Booker. I am President of Walter M. Booker and Associates, Inc., an incorporated biomedical group in Washington, D.C. I am also Professor Emeritus of Pharmacology at Howard University where I was Chairman of the Department of Pharmacology for 20 years.

My post graduate professional training consists of a Master's Degree in Cellular Physiology and Biochemistry from the University of Iowa and a Ph.D. in Physiology and Pharmacology from the University of Chicago. I also spent a postgraduate year as a Senior Fulbright Scholar in Belgium and Sweden, studying with two Nobel Laureates.

I hold membership in numerous scientific societies including the American Society of Pharmacology and Experimental Therapeutics, the American Physiology Society, the American College of Clinical Pharmacology, and the American College of Cardiology of which I am a Fellow.

The views I am expressing in this statement area based upon my training and experience as a scientific researcher in the fields of pharmacology and physiology. In these areas my research endeavors included studies on the effects and mechanisms of action of nicotine on the heart and the autonomic (sympathetic) nervous system.

My main concern with the Bills under consideration, HR 4957 and S 1929, is that they propose congressional action based on findings that have not been clearly established. It is important to note that both Bills make such summary statements as: "Cigarette smoking is the number one cause of lung cancer" and one-third of heart disease deaths "are attributable to smoking." While such opinions are undoubtedly based on some published information, there is by no means unanimity in the scientific community that cigarette smoking can be incontrovertibly labeled as causal.

Despite what those in the legislative arena might believe, the cause or causes of cancer of the lung (and other organs) remain unknown. Both smokers and nonsmokers contrast cancer and other diseases often associated with cigarette smoking, and we still don't know why.

The most accurate and appropriate statement that can be made is that cigarette smoking has been identified as one of the many risks or associated factors that may cause or aggravate certain commonly occurring diseases. We are still not able to say whether all relevant risk factors and associations have been identified and consequently, it is practically impossible to control for their effects in experimental studies. While attempts have been made to evaluate certain factors individually, research has not reached the point of being able to say a particular factor is causative.

The scientific fact of the matter is that no one knows why someone like an industrial worker develops cancer of the lung. Is it because he or she is a smoker or because he or she is an industrial worker? Should cigarette smoking be used as a "scapegoat" or should meaningful efforts be made to afford protection to the industrial worker?

Having devoted a large part of my research career to functional underlying processes that might contribute to certain diseases of the heart, I am particularly concerned with those parts of the Bills that seem to ask Congress to decide what specifically causes coronary artery disease. In all sincerity, I raise the question: If smoking has been scientifically shown to be a cause of heart disease, what are the established mechanisms? As a research scientist, I believe that such mechanistic data are necessary before conclusions on causation are possible.

Diet, whether deficiencies or excesses, lack of exercise, emotional stress, genetic factors, auto-immune factors (particularly where cancer is concerned) must each bear its responsibility as a possible cause of disease; but not one of these can be given sufficient responsibility to justify congressional action of the type contained in the Bills being discussed here today.

In summary, I strongly disagree with the proposed Bills because they misrepresent the present state of scientific knowledge. The Bills are asking Congress to give the Nation a scientific verdict on the causes of complex diseases when much of the evidence is either conflicting or has yet to be presented.

Statement

Smoking and Fetal Growth

by Oliver Gilbert Brooke, M.D. FRCP
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I am a graduate in Medicine of London University, attaining my basic medical degree in 1966. I received an MD degree (British medical equivalent of PhD) from London University in 1974. I am a pediatrician by training, with specialist interest in newborn medicine and nutrition. My present position is Reader in Pediatrics at St. George's Hospital (equivalent of full Professor in the United States) and I am head of Neonatology. Among other positions I have held, I have served as Scientific Officer of the Medical Research Counsel, Tropical Metabolism Research Unit at the University of the West Indies (1969-1972); Member of the Scientific Staff of the Division of Human Physiology, National Institute for Medical Research, Hampstead (1972-1973); Paediatric Registrar and Senior Registrar in Paediatrics at St. Mary's Hospital, London (1973-1976). I am a member of the European Advisory Committee on Feeding of Low Birth-weight Infants which was organized by the European Society of Paediatric Gastroenterology and Nutrition. In addition, I am a member of the following scientific societies: Nutrition Society, Physiological Society, Neonatal Society, British Paediatric Association, European Society of Paediatric Gastroenterology and Nutrition, and Paediatric Research Society. I have been an invited speaker and have presented numerous papers at scientific meetings, and I am the author of numerous scientific publications.

My interest in smoking and fetal growth dates from the early 1970s when I carried out a study on ethnicity and birth size. This involved careful matching of pregnant women for factors such as social status and income, combined with accurate assessment of gestation. Such information is difficult to obtain in large scale epidemiological surveys but is very important in assessing the outcome of pregnancy. When we analyzed our results we found that, after controlling for maternal age, height, gestation of pregnancy, parity, socio-economic status and race, and infant sex, smoking appeared to have little or no influence on birth weight and other measures of fetal growth. (Alvear J & Brocke OG, Archives of Disease in Childhood 53, 27, 1978; Brooke O.G., Lancet 1, 1158, 1977). Since this unexpected finding was at variance with the prevailing views I became interested in carrying out further research in the subject.

A review of the literature of the effects of smoking in pregnancy is contained in the U.S. Public Health Service report "Smoking and Health" (1979, 79-50066). This report marshals a large body of evidence to link smoking with low birth weight. However there is a strong minority body of opinion which holds that the effects observed in humans are mainly mediated through social status or nutrition. The hypotheses here are as follows:

1. Women of low socio-economic status have been known for many years to have smaller babies than more privileged women. Smoking is a class linked practice. There are now far more smoking women in lower than in upper social class groups in Western societies. The link between smoking and low birth weight may therefore not be causal but mediated through other social factors.

2. Smokers may be more likely than non-smokers to have small infants because of their personality or their genetic predisposition to respond unfavorably to stressful events.

3. The effect of smoking could be mediated through nutrition; it is possible that smokers eat less or worse than non-smokers, and that this is the cause of the association between smoking and reduced birth size.

Evidence in favour of one or another of these hypotheses has been obtained by a number of workers over the past 15-20 years:

Yerushalmy produced evidence that smoking was an "index to a particular type of reproductive outcome" (Yerushalmy J, Am. J. Obstet. Gynecol. 73, 808, 1957; Yerushalmy J, Am J. Epidemiol. 93 443, 1971).

Silverman's research supported these conclusions (Silverman D.T. Am. J. Epidemiol. 105, 513, 1977).

Davies et al, in a substantial and well documented study, concluded that much of the effect of smoking on fetal growth was mediated through poor maternal weight gain in pregnancy and was likely to have been of nutritional origin (Davies D P et al, Lancet 1, 385, 1976).

Rush reported similar findings to Davies in an equally well-conducted study (Rush D, J. Obstet. Gynaecol. Br. Cmmwlth. 81, 746, 1974). Rush also found that an effect of smoking on birth weight was confined to women of lower social class (Rush D., Am J. Dis Children 129, 430, 1975). More recently these findings have been confirmed by Papoz et al, who found no effect of smoking on birth weight in Parisian women of upper social class (Papoz L. et al, in: Maternal Nutrition in Pregnancy, Ed. Dobbing J. London: Academic Press, 1981).

I have been involved in research during the last two years to try to clarify these issues. This research has been supported by the American tobacco industry. It involves a team approach (epidemiological, obstetric, pediatric, nutritional and psychological) to the investigation of pregnancy outcome in a hospital providing maternity services to a typical urban community with widely varying socio-economic conditions. The initial 18 month period has been involved with a pilot study of an unselected group of 160 women, examining the feasability of employing various study instruments to evaluate social status, psychological health and nutrition. The results are in the process of analysis.

The next 2 years will, it is hoped, be devoted to answering questions about the relationships between smoking and food intake and between psycho-social stress and birth size. This will lead directly to a large scale epidemiological survey of pregnancy outcome with very careful attention to data collection techniques, and multivariate analysis of factors important in birth size and pregnancy outcome. The techniques used, particularly in relation to the collection of social and nutritional data, will have been thoroughly developed and tested in the initial studies.

March 8, 1982

Curriculum Vitae

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Present Appointment: Senior Lecturer in Child Health and Honorary Consultant Paediatrician. St George's Hospital Medical School, Cranmer Terrace, London, SW17

Medical Education: 1st and 2nd M.B., Kings College, London University.

M.B., B.S. (1966), St George's Hospital, London University.

Prizes: Brackenbury Prize in Medicine, St George's Hospital Medical School, 1965.

Other Degrees and Diplomas:
MRCP (London) 1968.
M D (London) 1973. Thesis title: "Malnutrition and Body Temperature in Jamaican Babies".
FRCP (London) 1980.

Previous Appointments:

1966-67 - House Physician to Medical Unit, St George's Hospital, London, SW1

1967-68 - House Surgeon to Mr Riddell, St George's Hospital London, SW17

- Senior House Officer to Neurological Unit, Atkinson Morley's Hospital, London, SW20

1968-69 - Senior House Officer to Infectious Diseases Unit, St George's Hospital, London, SW17

- Senior House Officer to Medical Unit, St George's Hospital, London, SW1

1969 - House Physician to Prof. Bywaters, Hammersmith Hospital and Royal Postgraduate Medical School.

1969-72 - Scientific Officer, Medical Research Council, Tropical Metabolism Research Unit, University of the West Indies, Jamaica.

1972-73 - Member of Scientific Staff, Division of Human Physiology, National Institute for Medical Research, Hampstead.

1973-74 - Paediatric Registrar, St Mary's Hospital, London, W2.

1974-76 - Senior Registrar in Paediatrics, St Mary's Hospital, London, W2.

Scientific Societies: Member of Nutrition Society
Physiological Society
Neonatal Society
British Paediatric Association
European Society of Paediatric Gastroenterology & Nutrition
Paediatric Research Society.

Other Scientific Activities: Member of European Advisory Committee on feeding of low birth-weight infants, organised by the European Society of Paediatric Gastroenterology and Nutrition.

Invited contributions at scientific meetings:

XIV International Pediatric Congress, Buenos Aires, 1974. Co-ordinator of Colloquium on Protein-Calorie Malnutrition, and invited speaker.

8th International Symposium on Paediatric Work Physiology, Bisham Abbey, 1976. Invited speaker.

National Conference on Development and Handicap, Nottingham University, 1977. Invited speaker.

Conference on Neonatal Medicine, Institute of Obstetrics, Queen Charlotte's Hospital, 1979. Invited speaker.

Royal College of Physicians Paediatric Conference, 1979 "Nutrition in Paediatrics". Invited speaker.

Nutrition Society Symposium on Nutrition in the Mother and Child, Royal Society of Medicine, 1979. Invited speaker.

Conference on the Biology of Development, Hôpital Necker, Paris, 1980. Invited speaker.

XVI International Pediatric Congress, Barcelona, 1980. Contributor to Symposium on Neonatal Nutrition

Annual Conference of Royal College of Radiologists, London, 1980. Invited speaker.

Papers presented at Scientific Meetings

1. "Influence of Malnutrition on central body temperature". Brooke, 1971. 16th Scientific Meeting of the Standing Advisory Committee for Medical Research in the British Caribbean, Barbados.
2. "Defective thermoregulation in malnourished Jamaican babies". Brooke, 1972. 17th Scientific Meeting of the Standing Advisory Committee for Medical Research in the British Caribbean, Nassau.
3. "Specific dynamic action in malnutrition". Ashworth, Brooke & Waterlow, 1972. 1st International Symposium on Energy Balance in Man, Paris.
4. "The metabolic rate of malnourished children". Brooke & Cocks, 1973. Physiological Society Meeting, Medical Research Council. Mill Hill, London.
5. "Evaluation of a method for measuring urine temperature". Brooke, Collins, Fox, James & Thornton, 1973. Physiological Society Meeting, Royal Postgraduate Medical School, London.
6. "Resting metabolic rate in malnourished children and its relationship with total body potassium". Brooke, 1974. Colloquium on protein-calorie malnutritic XIV International Pediatric Congress, Buenos Aires.
7. "High calorie feeding in protein-calorie malnutrition". Ashworth, Brooke & Wheeler, 1974. Colloquium on protein-calorie malnutrition, XIV International Pediatric Congress, Buenos Aires.
8. "Ethnic differences in neonatal anthropometric measurements". Brooke, 1975. Neonatal Society Meeting, Nottingham University.
9. "High energy feeding in low birth-weight infants". Brooke, 1975. Neonatal Society Meeting, St Thomas' Hospital, London.
10. "High energy feeding in immature infants" . Brooke & Alvear, 1976. 2nd European Nutrition Conference, Munich.
11. "Thermoregulation in Malnourished Children". Brooke, 1976. 8th International Symposium of Paediatric Work Physiology, Bisham Abbey,England.
12. "Influence of acidosis on energy absorption in immature infants". Brooke,1977. 48th Annual Meeting of British Paediatric Association, York. (plenary session).
13. "The role of the neonatal paediatrician in preventing visual handicap". Brooke, 1977. National Conference on Development and Handicap, Nottingham University.
14. "Postprandial energy expenditure in small-for-dates and immature infants". Alvear & Brooke, 1977. European Society for Paediatric Gastroenterology and Nutrition, Utrecht Meeting.
15. "Specific Dynamic Action in infants of low birth-weight". Alvear & Brooke, 197 Physiological Society Meeting, St George's Hospital, London.
16. "Postprandial metabolism in low-birth-weight infants". Brooke, 1978. 49th Annual Meeting of British Paediatric Association, York. (section on Nutrition and Metabolism).
17. "Energy cost of growth, in healthy immature infants". Brooke & Alvear, 1978. European Society for Paediatric Gastroenterology and Nutrition, Paris Meeting.

18. "Which small-for-dates infants are malnourished?". Brooke & Butters, 1978. Paediatric Research Society Meeting, Plymouth.

19. "Stools in infancy: frequency, weight, water & energy content". Lemch & Brooke, 1978. Paediatric Research Society Meeting, Plymouth.

20. "Circadian rhythms of melatonin and cyclic-AMP in neonates". Evans, Carter, Brooke & Smith, 1978. Paediatric Research Society Meeting, Plymouth.

21. "Effects of supplementary vitamin D during pregnancy and Asian women and their infants". Brooke, Bone, Brown, Carter, Maxwell & Robinson, 1979. 50th Annual Meeting of British Paediatric Association, York (plenary session).

22 "Energy balance in infancy". Brooke, 1979. Conference on Neonatal Medicine. Institute of Obstetrics, Queen Charlotte's Hospital, London.

23. "Energy metabolism in the neonate and malnourished child". Brooke, 1979. Royal College of Physicians Annual Paediatric Conference, Royal College of Physicians, London.

24. "Food and the fetus and neonate". Brooke, 1979. Nutrition Society Conference on Nutrition in the Mother and Child. Royal Society, London.

25. "Intestinal bypass in the rat: a model for Kwashiorkor?". Maxwell, McGowan & Brooke, 1979. International Conference on Infant Feeding and Diarrhoeal Disease, Kuala Lumpur.

26. "Lysozyme, IgA and carbonic anhydrase levels in the milk of mothers of preterm and fullterm infants". Brooke, Carter, West & Wood, 1980. Neonatal Society Meeting, Institute of Child Health, London.

27. "Changes in human fetal mineral metabolism associated with maternal vitamin D supplementation". Brooke, Brown & Cleeve, 1980. Biochemical Society Meeting, Surrey University.

28. "Energy balance in low birth-weight infants". Brooke, 1980. Conference on Biology of Development. Hopital Necker, Paris.

29. "Neonatal respiratory distress". Brooke, 1980. Annual meeting of Royal College of Radiologists, London.

30. "Protein concentrations in milk from mothers of preterm and term infants". Brooke, Carter, Hibbard, Wood & Brown, (1980). Biochemical Society Meeting, St George's Hospital, London.

Original Scientific Publications

1. Mechanism of action of β-adrenergic receptor blocking agents in angina pectoris.
Wilson, Brooke, Lloyd and Robinson (1969). Brit.Med.J. 4, 399.

2. Effect of ergotamine and ergometrine on forearm venous compliance in man.
Brooke and Robinson (1970). Brit.Med.J. 1, 139.

3. Iron absorption and pyrexia.
Beresford, Neale and Brooke (1971). Lancet 1, 568.

4. The influence of malnutrition on the post-prandial metabolic rate and respiratory quotient.
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5. Influence of malnutrition on the body temperature of children.
Brooke (1972). Brit.Med.J. 1, 331.

6. Hypothermia in malnourished Jamaican children.
Brooke (1972). Arch.Dis.Childh. 47, 525.

7. Thermoregulatory instability in kernicterus.
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8. A simple metabolism chamber allowing accurate determinations of evaporative water loss in large babies.
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9. Specific dynamic action in malnutrition.
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10. Fasting hypoglycaemia due to increased glucose utilisation and inappropriately high insulin.
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11. The metabolic rate of malnourished children.
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12. Evaluation of a method for measuring urine temperature.
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13. Response of malnourished babies to cold.
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14. Thermal insulation in malnourished Jamaican babies.
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15. Response of malnourished babies to heat.
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16. The importance of routine urine cultures in malnourished children.
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17. Resting metabolic rate in malnourished babies in relation to total body potassium.
Brooke and Cocks (1974). Acta.Paed.Scand. 63, 817.

18. Serum and liver triglycerides in malnourished Jamaican children with fatty liver.
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19. Measurements of deep body temperature from the urine.
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21. High energy feeding in protein-energy malnutrition.
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22.High fat feeding in immature infants.
Brooke and Alvear (1977). Nutr.Metabol. 20, Suppl. 2, 70.

23. Influence of acidosis on energy absorption in immature infants.
Brooke (1977). Arch.Dis.Child. 52, 809.

24. Specific dynamic action in infants of low birth weight.
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25. Postprandial energy expenditure in small-for-dates and immature infants.
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26. Fetal growth in different racial groups.
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28. Loss of energy during continuous infusions of breast milk.
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29. Energy retention, energy expenditure and growth in healthy immature infants.
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33. Effects of supplementary vitamin D during pregnancy in Asian women and their infants.
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35. Changes in human fetal mineral metabolism associated with maternal vitamin D supplementation.
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36. Food and the fetus and neonate.
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37. The ingestion by pregnant women of substances toxic to the foetus.
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38. Vitamin D supplements in pregnant Asian women: effects on calcium status and fetal growth.
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39. Energy metabolism in the neonate and the malnourished child.
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"Changes at Birth and Physiological Maturation in the Newborn Period and During later Childhood".
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Alvear and Brooke (1977). Lancet, 1, 1158.

Vitamin D deficiency in Asian immigrants.
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Brooke (1972). Brit.Med.J. 2, 164.

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"Human Growth" vol.3. (Falkner & Tanner).
Lancet 2, 775.

STATEMENT OF BARBARA B. BROWN, Ph.D.

I am Barbara B. Brown, former Chief of Experiential Physiology, Veterans Administration Hospital, Sepulveda, California. I received my Ph.D. in Pharmacology from the University of Cincinnati College of Medicine in 1950. I have over [illegible] years' research experience, primarily in the fields of pharmacology, psychopharmacology, neurophysiology and psychophysiology. I began my research activities as a technician in the pharmaceutical firm of The Wm. S. Merrill Co. (subsidiary of Merrill-Richardson) and later became Head of the Department of Pharmacology. After leaving Merrill, I served as Research Neuropharmacologist at Riker Laboratories in California and as Consulting Neurophysiologist at Psychopharmacology Research Laboratories. I was also an associate clinical professor of pharmacology at the University of California Center for Health Sciences in Los Angeles and at the Department of Psychiatry and Human Behavior at the University of California -- Irvine, and also lecturer at the Department of Psychiatry at UCLA. As Chief of Experiential Physiology Research at the VA Hospital in Sepulveda, I was one of the pioneers in the development of the concept of biofeedback. In the past few years, much of my professional activity has centered around attempting to analyze the role of mental activity (brain information processing systems) in health and illness. To date, I have published four books and several theoretical articles on this subject.

Given my extensive background in pharmacology, physiology, and behavior, I disagree with the conclusions and the basic generalizations of House bill 5653. This bill misinterprets the meaning of the reported statistical associations between cigarette smoking and certain diseases. It fails to understand the psychology of the multiple influences that determine individual lifestyles and the acceptance of their risks, and it completely ignores a fundamental and unresolved question: why do people smoke? Further, the bill misapprehends the level of public awareness of the widely publicized "health hazards of smoking," and it relies on the incorrect premise that revised warning labels could substantially affect that public awareness.

Smoking Behavior

Most important is the failure of the bill to address the many unknowns in smoking behavior. A great many perceptive people have observed that smoking behavior is a complex behavior determined by the interaction of several influences, namely, constitutional predispositions, environmental factors, experiential (psychosocial) influences, individual psychophysiologic reaction tendencies, and chemical factors. No single factor determines smoking behavior, nor do all interacting factors influence smoking behavior all the time.

The most convincing study ever conducted on the origins of smoking behavior was the decades-long study of constitutional predispositions to smoking by Seltzer. Using tests to develop psychological and physiological reaction profiles, Seltzer was able to predict who among pre-teens and young teenagers would be smokers when adults. His predictions were about 85% accurate.

Since a major objective of science is to predict events accurately, and Seltzer's work is an outstanding example of achieving this objective, it is unfortunate that the implications of his studies have not been actively pursued.

My own research did not come from considering Seltzer's work, but rather from a continuing interest in the electrical activity of the brain.

A report of my research study on smokers and human EEG activity appeared in the December 1968 issue of Neuropsychologia, an international journal on neurology. The study compared a variety of EEG measures, along with behavioral characteristics, among light, moderate, and heavy smokers, former smokers and nonsmokers. Each subject was studied one full day, under various control and test conditions, for EEG patterns and reactivity.

Results of the study were both unexpected and dramatic. I discovered striking differences between the brain wave patterns and brain electrical responses to sensory stimulation of heavy smokers as compared to those of nonsmokers. Results for the average smokers and for the former smokers were intermediate between the other two groups.

The outstanding characteristics of smokers' EEG patterns are a high frequency, rhythmic, high amplitude, sustained activity. It looks like alpha activity but is twice as fast. Alpha is commonly referred to as the brain wave correlate of relaxed wakefulness and is always contrasted with the nonrhythmic, low amplitude, very fast EEG activity that is commonly correlated with alert behavior. After comparing smokers' patterns with those of former smokers, with those of individuals in various emotional or physical states such as fatigue, etc., I decided that the best explanation for this distinctive EEG frequency is that it represents an inherent tendency for people who smoke to have a level of "alertness" higher than the average and also apparently excess brain electrical activity.

These absolute physiological findings can be interpreted in either of two ways: one, that the high frequency, rhythmic EEG activity is associated with information processing that is concerned with specific elements or thoughts, or that it is helter-skelter, poorly productive, non-linear mental activity. In either case, the smoker consistently shows an excess of (for lack of a better term) "cerebral" energy. "Cerebral" energy can be either productive or nonproductive, i.e., satisfy a predetermined objective or not.

I studied a small group of heavy smokers to learn whether their brain waves would show any effects from short periods of abstinence or any effects when they resumed smoking after abstinence. The heavy smokers who habitually smoked 2.5 to 5 packs of cigarettes per day were asked to refrain from smoking at least 12 hours prior to beginning the experimental recording session. We then intentionally prolonged the recording period in an attempt to increase the "need" to smoke, presumably making the EEG recording experience more stressful. After this, these heavy smoker subjects were allowed to smoke to satiation, and were allowed to smoke ad lib. throughout the remainder of the recording (about 2 more hours).

The results of this sub-experiment were quite surprising. First, no differences in brain wave patterns were found after the subjects had smoked to satiation as compared to patterns found after 12 hours of abstinence from smoking. Second, no changes in brain wave patterns were found at those times when the subjects were actually smoking cigarettes. And third, when the subjects were finally told that they could smoke after the abstinence period, only 1 of the 9 expressed any particular desire or need to smoke. One interpretation of this is that the novelty of the experimental situation acted as an effective substitute for smoking for this brief period of time.

The sustained and marked differences in brain wave activity between smokers and nonsmokers are very probably unrelated to the pharmacologic aspects of smoking. When nicotine is administered to animals, or when the effect of smoking is measured, a stimulant effect is only briefly noted. If the continuing action of nicotine and smoking, especially in "smoking" doses is a tranquillizing one, then the fast EEG patterns of the habitual smokers cannot very well be a result of their smoking activity. The remaining conclusion, then, is that there is a constitutional difference between people who tend to smoke and those who do not.

My studies, and those of Seltzer, comprise powerful evidence that constitutional factors can predispose to smoking behavior or some equivalent behavior that effectively diffuses the excess "cerebral" energy (such as the "high" that runners or joggers enjoy). This does not mean that shaping factors such as genes, home environment, individual experiences, etc., predispose specifically to smoking behavior, but that these factors predispose to a behavior that can discharge excess "cerebral" energy and at the same time be socially acceptable. In other words, people likely to smoke are people who have this particular kind of excess energy -- and smoking provides on acceptable outlet by which that energy can be discharged.

Smoking and Health Studies

The bill's "findings" on smoking and health reflect the government position which is promulgated in the Surgeon Generals' Reports. These Reports are based on highly selective reviews of that literature. The behavioral section of the 1979 Report, for example, admitted this very scientifically uncharacteristic approach. Many excellent "dissenting" reports are ignored. Frequently, when differing reports are cited, they are either inadequately or inaccurately reported. One wonders if occasionally the Reports' authors might not have been biased because of the government's policy on smoking. Unbiased reviews -- and unbiased research -- cannot be fostered in such an atmosphere.

Many research studies cited in the Reports contain serious errors, such as questionable criteria for selecting subjects, poor experimental design, weak criteria for evaluation of results, conclusions not warranted by the data, use of error-ridden or inconsistent public records for information on death and disease, and the abuse of statistics.

One of the weaknesses of these studies that most concerns me is the failure of the epidemiological studies to account for biological variation. In the behavioral area, this translates to the fact that the reasons for smoking differ in different kinds of people. The sum of psychological, constitutional, experiential, motivational factors is never the same for all smokers. There simply is no one, single condition of smoking behavior (incidentally, no single factor accounts for the high rate of recidivism in most stop-smoking programs). Moreover, studies that classify the one pack a day dilettante smoker (one who doesn't care whether he smokes or not, but does so for social reasons) along with the pack a day smoker who sincerely enjoys his smoking but limits it to a pack a day are extremely bad studies from the standpoint of subject selection.

Congress must remember that science is not infallible. Both scientists and scientific reports have normal distribution curves of quality. Very few are excellent; most are just average.

Awareness

In regard to awareness, I strongly disagree with the implication in House bill #5653 that the American public is still too ignorant of the hazards that have been associated with cigarette smoking. I can think of no health claims in the past 20 years that have received more intensive and pervasive media coverage and governmental attention. The media regularly cover all research on smoking and health, particularly reports that suggest hazardous effects. This often amounts to a media blitz.

Further, official warnings are constantly reinforced by anti-smoking commercials and advertisements on radio, magazines, and TV. Advertising by stop-smoking institutes, courses, clinics, etc., constitutes additional media coverage and anti-smoking campaigning. Finally "educational" programs are a fairly permanent part of many school programs. A recent TV news report showed first graders signing oaths that they would never smoke and orally repeating the oath virtually every day. Such a strong effort, which is probably representative of many educational programs, goes beyond merely an education purpose and approaches behavior or thought control.

Warning Labels

Even if I agreed with the bill's findings and supported the belief that public awareness of health hazards was inadequate, I would still disagree with the proposed charge in health warnings. As a society, we are extremely uninformed about the basis for behavior that we have termed risk-taking. It should be obvious that the extraordinary sums of money spent on behavioral research, its almost complete failure to discover the causes of these behaviors, and its near failure to prevent such behaviors carry a message. One of the outstanding flaws is the failure of scientists or interested people to encourage studies of personality factors (not neuroticism scores which relate more to neuroses than normal behavior), studies on the role of the environment, studies on the role of psychosocial influences (again the normal rather than the abnormal ones), studies on the role of the human intellect on behavior, and the role of everyday stress on smoking behavior.

Congress must understand that even the so-called experts just don't know enough about why people behave the way they do in a variety of areas, and how that behavior relates to the individuals' health. We need for example tests built on the characteristics of normal people; we must study the normal range of behavior, rather than the abnormal. The majority of smoking behavior studies have used conditioned learning theory. Conditioned learning is an automated, primitive kind of behavior (obviously, since most of the studies are done in rats and pigeons). This theory is unable to explain a behavior as complex as smoking.

Smoking habits have changed, but only a minute part of that change is due to anti-smoking campaigns. People likely to smoke may be people who have excess "cerebral" energy, and our society in recent years has provided them with a variety of other acceptable outlets for that energy. Before any governmental program to reduce cigarette smoking should be considered, a total reevaluation and restructuring of research into smoking motivation and behavior should be commenced. Because of my research, I feel strongly that the reasons people choose to smoke have constitutional origins and this must be taken into account when the advisability of a government cessation campaign is discussed.

Conclusion

House bill 5653 should not become law, because its scientific underpinnings are weak and unconvincing.

Statement of Dr. Victor Buhler

My name is Victor Buhler and I am a pathologist living in Kansas City, Missouri. I am certified by the American Board of Pathology in both pathologic anatomy and clinical pathology. I have held faculty appointments at the University of Kansas School of Medicine and the University of Missouri-Kansas City School of Medicine. I have served as the President of the College of American Pathologists, the Missouri State Medical Association, the Missouri Society of Pathologists and the Kansas City Society of Pathologists. I am a Fellow of the American Society of Clinical Pathologists, the American College of Physicians, the American College of Pathologists, and a member of the International Academy of Pathology and the American Association for the Advancement of Science. I participated upon invitation by President Johnson in the White House Conference on Health in 1965.

At the present time, I am associated with a group of pathologists serving one major hospital and several community hospitals in the Kansas City area, with primary responsibility for the Liberty Hospital, Liberty, Missouri.

I have become familiar over the years with many of the articles cited in the various Surgeon General's reports on smoking and health and with the general medical literature pertaining to this subject. I testified before the Committee on Interstate and Foreign Commerce in 1969 in regard to proposed legislation concerning cigarette labeling and advertising. At that time, I told the Committee that my knowledge of the medical literature, my own investigations and my laboratory experience established "my firm conviction that neither cigarette smoking nor any other etiological agent has been shown to be the cause of cancer of the lung." I have seen no research reports in the intervening 13 years which would change that view. I am pleased to have the opportunity to give you my views on the present legislative proposals embodied in S. 1929 and H.R. 4957.

I am most disturbed by the finding included in both of these legislative proposals that cigarette smoking is the major cause of lung cancer. Such a statement is, in my view, not supportable by the pathological and clinical observations I have made during my practice, nor by my review of the medical literature. I am disturbed to see such findings in proposed federal legislation because I believe it is deceptive and misleading to the public and the medical community. The pressure to find causes and cures of cancer and other chronic diseases is considerable, and it should be. The problem, however, is that this pressure creates great temptation to seize upon easy answers. It is always important to realize that objectivity is vitally necessary for scientific progress. The cause or causes of lung cancer are unknown, and a Congressional finding to the contrary does not alter that situation.

Our only hope for progress in the fight against this dread disease is in innovative medical research, not legislative pronouncements.

My view that cigarette smoking has not been proven to be a cause of lung cancer is based on my personal knowledge of the sources for the epidemiological evidence frequently used to support this theory. Epidemiological studies, for the most part, are based on mortality data derived from statements of physicians or others who sign death certificates. In approximately 80 percent of deaths, no post-mortem examinations are done to verify the information on the death certificate regarding the primary or underlying causes of death. Even when autopsies are performed, the results often are not available until after the death certificates have been completed. The recorded causes of death, then, are primarily based on the clinical diagnoses, and it has been my experience as a practicing pathologist that these are frequently incorrect. If death certificates reflected the results of completed autopsies, the statistics would have a much higher degree of validity. At the present time it must be understood that 80 percent or more of all death certificates cannot be considered as having been scientifically validated. Consequently, I have grave doubts about the statistical associations which have been derived from data which is subject to serious flaws.

In addition, the data in statistical studies of lung cancer do not distinguish between primary lung cancer and secondary lung cancer. Primary lung cancer is a malignant tumor originating in the lung. Secondary lung cancer, often called metastatic, is cancer that has spread to the lung from another body site. I have all too frequently found in my own work that tumors clinically considered as primary in the lung are often metastatic and result from cancerous growths in other parts of the body.

One or the reasons why primary lung cancer may be frequently diagnosed when it is not present is that physicians depend primarily on chest x-rays as the technique for diagnosing it. In this regard, it should be noted that chest x-rays present fewer problems in interpretation than x-rays of any other organ or part of the body except the skeletal system. This is because the air in the lungs provides an excellent contrast medium so that disease states are more readily detected. We find, therefore, that it is easier to observe on x-ray an abnormality in the chest rather than, for example, the liver. Even then, however, one cannot always tell from an x-ray whether an area of density in the lung is a nonmalignant or malignant growth, and if malignant, whether or not it originated in the lung.

There are of course, techniques other than x-rays for diagnosing lung cancer. Biopsy and sophisticated scanning techniques (CAT scan), two other routinely used methods, do not always allow for distinguishing between secondary and primary tumors. It should be stressed that primary lung cancer is simulated by secondary tumors arising from other organs of the body with sufficient frequency to challenge the diagnostic accuracy of many deaths certified as primary lung cancer without autopsy exclusion of other primary sites.

In my judgement, the failure of epidemiological studies to distinguish between primary and secondary lung cancer in their statistical analyses raises serious questions about causal interpretations of statistical associations.

Some advocates of the smoking causation hypothesis have said that lung tissue from smokers exhibits certain abnormalities that they describe as "pre-cancerous." They conclude then that cigarette smoking causes these changes which eventually will lead to lung cancer. Let me say first of all that based on my examination of thousands of lungs in microscopic detail that no one can determine whether or not a lesion described as "pre-cancerous" will progress into cancer.

Metaplasia (changes of one type of tissue to another type), hyperplasia (increases in cell numbers), and dysplasia (atypical cytologic changes in cells) can be seen in lungs of both smokers and non-smokers. All of these kinds of changes are rather common and should not be considered as pre-cancerous. In fact, the weight of scientific evidence is that these lesions will not progress to cancer. Metaplasia, common in older individuals, occurs frequently in the trachea or windpipe, and yet tracheal cancer is extremely rare. Any study of autopsy cases that suggests that metaplasia, hyperplasia, or dysplasia provides evidence that smoking leads to lung cancer oversteps scientific bounds. Through my own experience, I cannot confirm that significant cell or tissue changes in the lung occur more frequently in smokers than in non-smokers. Certainly there are smokers without changes, and non-smokers with changes.

As a pathologist, I find the arguments on "pre-cancerous" lesions to indict smoking as a cause of lung cancer to be unconvincing and indeed inconsistent with my own clinical observations.

I might add that, contrary to the reports that one often hears, it is impossible for the pathologist to determine from both gross and microscopic examination of lung tissue whether the person is a smoker or a non-smoker.

I am equally unconvinced by the evidence provided by animal experimentation. As the Congressional Record shows, I twice described to Congressman Tim Lee Carter the critical weaknesses in one of the few inhalation experiments that have supposedly produced cancer -- the Auerbach beagles study. That experiment suffered from severe design defects, and the photomicrographs published with the article would not permit most pathologists to reach the conclusions stated by the authors. I described to Congressman Carter the essential failure of tobacco smoke inhalation experiments to induce lung cancer in animals. That comment is still valid today: no inhalation studies have shown that tobacco smoke inhalation causes lung cancer. Other experimental methods of exposure, such as skin painting, fail to realistically approximate actual human exposure, so that the results of these experimental models must be viewed with suspicion.

Another important aspect of the smoking and lung cancer question involves the histological (cell type) classification of tumors. Some have suggested that the newer cigarettes, generally those with lower tar and nicotine yields, are responsible for the emergence of different frequencies of the various cell types. Squamous cell carcinoma is the type of cancer that has generally been associated with cigarette smoking. It is less frequently found in non-smokers and in woman, for reasons that are not yet known. Recently, a few reports have suggested that the relative frequency of squamous cell cancer is decreasing, while that of adenocarcinoma, the type formerly found more commonly in women and non-smokers, may be increasing. I find the interpretation that these new patterns are caused by recent changes in cigarette manufacture to be highly questionable.

First, I believe that the few isolated reports lack sufficient information about such rudimentary factors as nutritional status, occupational exposure, or ethnic background. Consequently, they do not provide, in my view, a reliable measure of the changes in the general population.

Further, of a more technical nature, but of cardinal importance to the pathologist, is the cellular variation produced by the different methods of specimen procurement, fixation, storage, and staining techniques. In addition, the reported frequency of certain cell types will depend upon the source of the specimens; sputum cytology, for example, yields very different results from specimens taken at surgery, which in turn differ from those taken at autopsy.

Most important of all, however, is the fact that there seems to be virtually no solid evidence to link cigarettes with these reported changes. Until more rigorous scientific standards are applied, this change in cell type frequency, if it is real, remains unexplainable and of unknown significance.

Based on my years of experience as a pathologist and on my reviews of the smoking and health literature, I must disagree strongly with the proposed Congressional finding that cigarette smoking is the major cause of lung cancer.

Victor B. Buhler, M.D.

STATEMENT OF

JACK MATTHEWS FARRIS, M.D.

My name is Jack Matthews Farris. I am Emeritus Professor of Surgery at the University of California at San Diego. I practiced surgery in the Los Angeles area for more than 25 years as Staff Surgeon at the Good Samaritan Hospital, Los Angeles; Chief of Surgical Service at the Harbor General Hospital, Torrance, California; and Chief of Surgical Services at the California Hospital Medical Center, Los Angeles. I also served as Senior Consultant to Surgery for the Veterans Administration, for whom I directed the surgical activities at several of their institutions in California.

I am on the Board of Directors and Board of Trustees of the Scripps Clinic and Research Foundation, and was on the Board of Directors for the Medical Research Association of California. I am a member of numerous medical societies where I have served on advisory committees. I have published approximately 73 papers in scientific journals and have contributed several chapters for surgical textbooks. My full Curriculum Vitae and list of publications are submitted with this statement.

Throughout the past 25 years, I have followed with interest the evidence for and against the thesis that cigarette smoking has a causal relationship with cancer of the lung. This interest has been stimulated by my operation on more than 8,000 patients, many of whom suffered from cancer in various parts of the body, including lung cancer.

In 1965, I submitted statements to the House of Representatives and the Senate in which I said that I did not believe that lung cancer had been shown to be caused by cigarette smoking, and that any conclusion to the contrary was not justified.

My opinion today remains firmly the same. There are numerous complexities in the behavior and cause of cancer in all parts of the body. I believe that, when we learn how and what causes cancer, we might well find that cigarette smoking has little or nothing to do with the genesis of carcinoma of the lung.

That the smoking and health controversy continues is demontrated by many studies and findings that cannot be explained by a smoking causation hypothesis. I would like to mention just a few of these.

1. Persons who have never smoked get cancer of the lung that is indistinguishable from those cancers that are reported in smokers. A recent study has shown a significant and increasing incidence of cancer of the lung in non-smokers.

2. In the early 1960's researchers, without regard to smoking in the population, predicted that the death rate in lung cancer would level off in the next decade or so. Studies since then have supported their prediction.

3. Cancer is rarest in those parts of the body most in contact with cigarette smoke, for example, lip, tongue, trachea and larynx. Tracheal cancer continues to be an extremely rare disease, even though the trachea, which is part of the respiratory tract, is more exposed to tobacco smoke than the lung. The larynx is also more exposed to tobacco smoke than the lung; nevertheless, the incidence of laryngeal cancer has remained relatively stable during the last quarter century and has not followed the reported increase in lung cancer.

4. Lung cancer often originates in areas of the lung that have little or no contact with tobacco smoke, that is, in the peripheral areas of the lung, rather than in the hilar, or central, region.

5. Although there is no reason to suppose that there is greater exposure of tobacco smoke at the site of any given cancer in the lung, lung cancer usually appears as a solitary lesion. Metastatic cancer, on the other hand, such as that which spreads from the breast, may produce hundreds or thousands of minute lesions in the lung field. Moreover, once a lobe containing cancer of the lung has been removed, a second primary cancer of the lung rarely develops, although the lung fields have been exposed to the same amount of inhaled smoke.

6. The vast majority of heavy smokers don't get lung cancer. One theory that is being investigated is that the immune system has a lot to do with the development of this disease. —

7. Efforts have failed to produce the kind of lung cancer reported to be statistically associated with smoking in tests on animals exposed to smoke. One study has shown that a group of hamsters exposed to cigarette smoke lived longer than those not exposed.

8. Some studies find that in workers exposed to certain chemicals the smokers have a lower incidence of cancer than the non-smokers. For example, this has occurred in workers exposed to chloromethyl ethers.

It is clear that the smoking and health controversy has not been resolved. It is also clear that cigarette smoke cannot be the total answer to cancer of the lung.

All of us who are interested in this controversial subject, have one thing in common - we are all anxious to see a discovery for the prevention of cancer, which inexplicably claims so many lives today. The problem won't be solved by legislating the cause of disease. The problem can only be solved by continued exploration of the various theories and factors suggested by the research to date.

STATEMENT OF SHERWIN J. FEINHANDLER, Ph.D.

I am Dr. Sherwin J. Feinhandler, President of Social Systems Analysts, Inc., a social and behavioral research and consulting firm, in Watertown Massachusetts. I am a consultant and have run projects for federal, state and local government agencies as well as educational institutions and organizations in the private sector. By training I am a Cultural Anthropologist with degrees from the departments of Sociology at Northwestern University, Anthropology at Syracuse University and Social Relations at Harvard University. I have been Assistant Professor in the Department of Psychiatry at Boston University School of Medicine and Assistant Professor in the Department of Anthropology at Boston University. More recently I have been a Lecturer in Anthropology in the Department of Psychiatry at Harvard Medical School.

My research activities have included a focus on social and cultural processes in everyday life, communities and organizations. I have published articles and presented papers on various topics related to these areas. Among my interests is the role of and reactions to tobacco in society. Therefore, I was quite concerned when I heard of proposed House Bill 4957.

The following statement is based upon data collected over several years from smokers, ex-smokers, nonsmokers and anti-smokers on behavior and meaning systems related to tobacco and its use. The data were generated through:

- intensive interviews with people representing all ages, both sexes, and a variety of ethnic, educational and social class backgrounds;
- intensive and extensive group discussions with the same representative segments of the population;
- structured observations of behavior of smokers and nonsmokers in everyday life;
- and recent interviews focusing on decisions made by smokers in relation to their understandings of health claims and smoking.

In our earlier studies we found that smokers and nonsmokers alike were aware of and generally accepted the claims concerning detrimental consequences of smoking. We learned that smoking occurs as customary patterned behavior and has powerful support in both personal and social needs which cannot be readily abridged without serious consequences. Our purpose in conducting new interviews was to determine whether smokers make decisions about smoking based on their awareness of smoking related health claims and warnings. If so, what is the process and what are their reasons for deciding whether or not to continue smoking. In the course of this investigation several issues emerged in relation to these questions. These will be described below.

AWARENESS OF HEALTH CLAIMS

The Surgeon General's reports and the related coverage of their findings have been extremely effective in communicating messages about health and smoking to the public. Our respondents made it obvious that they were keenly aware of the warnings about smoking, usually without our raising the question. When asked her views about the relationship of smoking to health, one respondent's first words were: "The Surgeon General says; 'and whatever he says', smoking is bad for you, and I know it".

Our studies show no tendency on the part of smokers to reject the idea that smoking can be hazardous: "I am convinced it is a problem". For some it is a highly salient one: "I think about it all the time". Even when the issues are less salient for smokers, their concerns are deeply felt. Clearly, smokers know the claims about their smoking, and some attribute a degree of certainty to its consequences which may not be in technical agreement with the facts: "I think (smoking) gives you cancer. What else can I say about it".

Smokers perceive the consequences of smoking to be real, and often imminent even though they themselves feel no negative effects. "Smoking doesn't seem to hurt me. Maybe it is because I have not smoked long enough (to notice any effects)".

Responses to Warning Labels

In the face of this general awareness, our respondents treat health warnings on cigarette packs and in ads as self-evident. When asked about awareness of the Surgeon General's warning on every cigarette package, a respondent commented: "All we smokers know smoking is bad". In many instances the warnings are found to be somewhat insulting to the intelligence and raise issues of the motivation behind these attempts to inform. As a consequence, the credibility of the health issues is questioned.

When confronting a situation involving risk, most people engage in a kind of risk assessment in which they attempt to weigh jointly the seriousness of the consequences and the probability of their occurrence. The question of credibility is especially relevant here, since it is raised by some smokers in relation to specific health claims.

Specific statements often raise credibility issues when people have contrary evidence; they then lower their assessment of the probability of risk. They are aware of the specific health claims but disbelieve them.

When asked how they would respond to a warning concerning the relationship between lung cancer and smoking, typical responses of this type were:

> "I don't feel shortness of breath ... I know of lots of cases where people who never smoked got lung cancer and there are so many smokers who don't get it ..."

> "Changing the warnings -- it would scare but then again I laugh because its just not true. Everyone who smokes doesn't get lung cancer. It is just not enough to give up. Maybe I am just stubborn but I don't like being told what to do. The thing that would make me give it up is my own feelings, what it does to my own body."

A more common response among smokers involves some kind of acceptance or belief in the health claims. It is these smokers who engage in a more complex process of deciding whether or not to smoke.

INFORMED DECISIONS TO CONTINUE SMOKING

The fact that smokers continue to smoke, although aware of the claims concerning the health consequences of smoking, may lead to the conclusion that they are engaged in an irrational process. This process has often been dismissed as mere habit. Quite the contrary, we have found that smokers engage in a conscious decision process, not only about whether to continue smoking, but also about whether or not to smoke certain cigarettes through the day.

We have been struck by the general similarity of the process used by people from quite different backgrounds when health considerations are the stimuli for reassessing the decision to smoke. We have described above the approach used by people who question the risk of smoking. Others consider its risks and benefits; we have noted two different approaches. One involves the balancing of two sets of risks: that of smoking against that of not smoking. The other weighs the perceived benefits of smoking against the perceived risks of smoking.

The first approach involves weighing one or more of the predicted consequences of not smoking against the predicted consequences of smoking. The smoker assesses the probability of the consequences on both sides of the choice and jointly determines benefit in terms of choosing which risks are preferred.

> "I tried to give up smoking 4 or 5 times and went maybe three months and put on like 20 to 25 pounds ... for my own health I went back to smoking."

> "I've tried not smoking and found these days to be laden with anxiety. With that choice, I prefer smoking."

The second approach defines the benefits of smoking in terms of the value added to smoking by the uses to which it is put in achieving certain desired ends.

The Sources of Value

We have observed the use of tobacco in fulfilling a wide variety of personal and social needs. Smoking has value for people because of this role.

Lighting a cigarette in response to pleasant or enjoyable experiences, such as the company of friends or at the end of a good meal, is cited frequently. In this instance smoking is said to heighten the experience and enhance good feelings. The pleasure and relaxation many associate with the use of tobacco often derives from the sensorimotor aspect of smoking: taste, handling, visualization of smoke and inhaling and exhaling. One respondent explained:

> "If I really desire a cigarette nothing can replace it. Its not the nicotine. Its the feeling of the smoke going up and down that I enjoy. It is a unique experience. A pleasurable feeling."

People feel that smoking serves to reduce the emotional consequences of negative situations -- when experiencing feelings of fear, anxiety or distress. One informant characterized cigarettes as "a constant companion I carry for comfort."

Smoking plays an important part in the everyday ordering of individual lives. Smoking after a meal is a common practice, witnessed everyday in American society. "I normally stop eating, light a cigarette and then I know that mealtime is over. If I don't have a cigarette, then I feel compelled to eat at least one more thing." Here the act of smoking serves to mark the end of an event. As a structuring mechanism, the act of smoking brings with it a sense of predictability and familiarity which allows individuals to achieve a sense of security.

Smoking serves to impose structure on events which appear to have little or no structure of their own. "I smoke when I have to wait. Its something that helps me get through the waiting."

Cigarettes can aid in the organization of thought. "If I am writing I will stop and have a cigarette to collect my thoughts." Smoking is also viewed as a break from activities, a time in which to relax. "When I work steadily in one place for a long time, smoking is like a little rest period. A five minute break from what I am doing."

Smoking can serve as a social lubricant by providing a common activity for establishing social relationships. In our society the act of offering a cigarette or a light is an expression of a desire to be sociable. Smoking can also serve to affirm social bonds for individuals and groups whose relationships have been previously established.

> "I started smoking in college because there was a real division between smokers and nonsmokers. I was very shy and at certain times after dinner for example people would get together in the smoking room. It was my way to be accepted by these people and to make new friends. And I made a lot of friends through smoking. In the smoking room there was a feeling of cameraderie. You would talk to people that you wouldn't normally. Its a very clear thing, you meet a lot of people through smoking. It was easier to meet smokers than nonsmokers because you had something in common -- the vehicle of smoking."

Smoking is a behavior that a smoker shares with a wide sector of society. The behavior is patterned and the understandings are shared.

VIEWS OF THE PROPER ROLE OF GOVERNMENT

We have illustrated smoking as patterned behavior carried out separately by individuals over space and time, reflecting tradition and organizing concepts in common. Smoking, deeply ingrained in our culture through centuries of tobacco use, has thus become an accepted custom in society. This explains why the majority of smokers and nonsmokers share similar views about the government's role in relation to smoking.

These views can be summarized in terms of the social value placed on freedom of individual choice and the absence of arbitrary regulation when social processes can work out the issues at hand. There is also a feeling that externally imposed regulation removes responsibility from individuals and natural social groups. While some people rather like the idea of avoiding responsibility, they hesitate in agreeing to the inevitable sacrifice of free choice.

Most respondents feel that the appropriate role for government in relation to smoking is that of keeping people informed.

> "I feel that the government has the responsibility to warn people because no single person can accumulate all knowledge and conduct tests. I feel that government should conduct studies, I just don't feel that the government has the right to make your choice. Telling you you can't smoke is like telling you, 'you can't have an alcoholic beverage'. No attempt to do

> this would work, is silly and wastes a lot of money. All it does is to threaten the credibility of the laws of our country. Like prohibition, it would create a black market. It becomes common practice to break the law. It would make law-breakers out of people that are otherwise considered good citizens. Cigarettes are even less dangerous than alcohol because they don't have the potential to create deviant behavior."

The Attribution of Deviance

Current attempts to regulate the smoking process are seen as part of an effort to define smoking as deviant behavior. Many people recognize that the attribution of deviance is extended to the smoker himself.

> "I began to smoke at 24; there was nothing terrible about it then. There was no publicity. I can hardly enjoy it now. I feel very weak minded."

> "It seems to me that people are now trying to make moral judgements on smokers and the government is backing them. They try to put guilt on the smoker and it is the smoker who suffers."

Noteworthy in this respect are recent efforts to encourage businesses to discriminate against smokers in hiring and the establishment of a psycho-diagnostic category, "smoking behavior disorder".

Anti-smoking groups and government are felt to be active in this effort. Smoker and nonsmoker alike view the efforts of anti-smoking groups as deviant rather than viewing smoking as such. They question the motivation of people in these groups and attribute to them either Messianic or power seeking desires. Government attempts to restrict customary behavior are similarly questioned. People thus feel that government demonstrates hypocrisy, inappropriate exercise of power and undue manipulation of individual rights.

> "I really think that the government and the people who are anti-smokers like to point fingers. It makes them feel good. 'I'm not doing that, therefore I must be better than they are. I must be a better person'. There is no difference between smokers and nonsmokers. To categorize smokers or nonsmokers is ridiculous. We have enough discrimination in the U.S. now. Why try to create more. But it is anti-smokers against the rest of the world."

> "I think that this is all political. I think that there are people out there who are violently anti-smoking, not for health reasons and they have money

> and a lobby. If it was for health reasons they would be on the bandwagon for a hell of a lot of other things. People are bothering me and it is a personal insult. They shouldn't be allowed to bother me. Anti-smokers have no personal consideration or sensitivity. It is a legal way for them to relieve a lot of aggression. I may hate a woman wearing strong perfume but I don't pour water on her or tell her to get off the elevator because she is bothering me. There has to be something more going on. Its looking only after themselves; I am good and they're bad."

Most people reject the attributions of deviance applied to smokers because they recognize that smoking is part of customary behavior and place a high value on free choice in matters of custom. Many nonsmokers, even those who have experienced discomfort as a result of others' smoke in specific situations, have said that interpersonal processes are to be preferred to legislative restrictions.

SUMMARY

Smokers are keenly aware of the publicity surrounding the relationship between health and smoking and the warnings on cigarette packs. Given this awareness, they show no tendency to reject the idea that smoking can be hazardous, whether or not they have any desire to give up smoking. The sheer volume and intensity of the warnings has led many to question the motivation behind them, and the validity of their content. Specific warnings encourage smokers to consider easily found counter-examples.

This kind of behavior is often dismissed as irrational, however, it is a highly logical response to social needs and realities. Smoking is an indispensable aid in encouraging positive social behavior, and enabling individuals to deal with situations in the world about them.

The fact that people continue to smoke in the face of seemingly overwhelming publicity against smoking is proof, in itself, of its social importance. It follows that attempts, which smokers perceive to be unjustifiable coercion and hypocritical at best, involves social consequences whose costs are incalculable.

S.J. Feinhandler, Ph.D.

RESUME

Name: SHERWIN J. FEINHANDLER

Citizenship: U.S.

Specialization: Cultural anthropology; linguistics; language & communication; cognitive & meaning systems; community mental health; tobacco, alcohol & drug research; program evaluation; ethnography of everyday life; Africa, United States

Education:

Harvard University	9/58-12/67	Ph.D., Social Relations
Makere College (East African Institute of Social Research)	7/60-12/62	Associate
London University	7/60-12/60	Special Student
Syracuse University	9/56-6/58	M.A., Anthropology
Northwestern University	4/53-6/56	B.A., Sociology

Languages:
French: speaking, reading
German: speaking, reading
Swahili: fluent
Kikamba: speaking

Employment and Research Experience:

Dates	Title	Institution	Responsibility
2/70-pres	President	Social Systems Analysts, Inc.	Administration, research and consulting
1/70-6/80	Lecturer in Anthropology	Harvard Medical School Dept. of Psychiatry	Research in verbal and non-verbal behavior
1/70-6/80	Assistant Anthropologist	Dept. of Psychiatry Mass. General Hospital	Coordinator of Alcoholism Training Program '70-'71, consultant to Street Youth Program '72-'73, consulting '70-'80
9/72-6/77	Lecturer	Simmons School of Social Work	Research Seminars
1/70-6/73	Adjunct Assistant Professor	Dept. of Anthropology Boston University	Teaching
6/68-12/69	Assistant Professor	Division of Psychiatry B.U. School of Medicine	Director of evaluation of a housing program
9/67-12/69	Assistant Professor	Dept. of Anthropology Boston University	Teaching

Employment and Research Experience (cont'd):

Dates	Title	Institution	Responsibility
1/68-5/68	Research Associate	H.S.G. Cutter Brockton V.A. Hospital Brockton, MA	Research on alcoholism and communications
3/65-12/67	Research Assistant	L.J. Sherman Brockton V.A. Hospital	Research on alcoholism and hospital social structure
1/61-11/62	Ethnographer	East Africa C.O. Frake, Supervisor	Research, primarily cognitive anthropology, sociology, social structure and medicine
9/59-6/60	Research Assistant	R.F. Bales Harvard University	Research on interpersonal behavior
1/59-6/60	Researcher	M. Orne, Mass. Mental Health Center	Field study of Penecostal churches
1/59-9/59	Research Assistant	M. Orne, Mass. Mental Health Center	Experiments on trance
10/57-4/58	Researcher	N.Y. State Mental Health Research Unit, Syracuse	Research in schizophrenia
10/56-6/58	Instructor	Syracuse University	Teaching
10/56-12/57	Researcher	G. Agogino	Archeological Digs
12/54-4/55	Statistical Assistant	R.F. Winch Northwestern University	Data Analysis

Consulting Experience:

Dates	Client	Responsibility
9/76-10/78	Philip Morris, Inc.	Historical and structuralist studies on tobacco
8/78-9/78	Tobacco Institute	Consultant on tobacco legislation
11/73-2/74	National Institute on Alcohol Abuse and Alcoholism	Study of bar behavior
6/73-10/73	Mass. Committee on Criminal Justice	Evaluation of delinquency prevention practices
6/72-6/73	Cross-National Drinking Practices	Survey of drinking practices
9/72-4/73	Commonwealth of Massachusetts	Member of Governor's Advisory Committee on Drug Abuse Research

Consulting Experience (cont'd):

Dates	Client	Responsibility
3/72-12/72	Mass. Committee on Criminal Justice	Evaluation of methadone day-care program
3/71-9/72	Division of Drug Rehabilitation, Comm. of Mass.	Evaluation of Marathon House, a drug rehabilitation program
4/71-6/72	Beth Israel Hospital	Department of Labor study of job improvement
5/71-7/71	Boston University School of Medicine, Division of Psychiatry	Proposal for comprehensive treatment program for alcoholism
4/70-12/70	Harvard University Program on Technology and Society	Designed and conducted survey of public attitudes on technology
9/70-8/71	Barss, Reitzel and Associates	Consultant, Office of Education library program evaluation
11/68-10/69	Harold Cabot Co.	Studies of consumer cognition and behavior
1/67-6/67	Boston State Hospital	Design of treatment and research in alcoholism
5/66-7/66	Harvard University School of Education	Curriculum development on East Africa
3/65-9/65	Charles MacArthur Harvard University Health Services	Study of smoking, behavior and cognition

Professionally Related Experience:

Leading therapy group of alcoholics
Leading therapy group of wives of alcoholics
Leading therapy group of married couples
Leading "T" group of Psychology Trainees (graduate students)
Evaluating referrals on closed circuit Telediagnosis System
Curriculum development
Assistant Editor, The Deltan

Courses Taught:

Syracuse University:
    Introductory Anthropology
    Introductory Sociology
    Introductory and Intermediate Statistics
    Demography

Courses Taught (cont'd):

Boston University:
- Folk Systems of Classification
- Primitive Religion
- Language and Culture
- Medical Anthropology
- Anthropological Linguistics

Simmons School of Social Work:
- Research seminars

Antioch Institute of Open Education:
- Program Evaluation

Grants and Fellowships:

Fellow, Social Science in Medicine, Harvard University, 9/58-1/61
Ford Fellow, African Field Research, 1/61-6/62

Professional Memberships:

- Fellow of the American Anthropological Association
- Fellow of the Royal Anthropological Institute
- Fellow of the Society for Applied Anthropology
- Society for Ethnomusicology
- International African Institute
- Society of Medical Anthropology
- Council on Anthropology and Education

Publications:

1957 Amaranth Seeds from a San Jose Site in New Mexico. Texas Journal of Science 154-156. (with G. Agogino)

1957a Further New Concepts in Family Sociology. Deltan, Spring. (with L. Freeman, G. Agogino and N. Goldman)

1959 Social Class and Mental Hospital Prognosis. American Sociological Review 24:815-821. (with R. Hardt)

1969 An Evaluation of the Boston Rehabilitation Project. Monograph submitted to the Department of Housing and Urban Development. To be published as a government monograph.

1971 On the Training of Leaders in Alcoholism. In Recent Advances in Studies of Alcoholism. edited by J. Mendelson and N. Mello. Rockville: N.I.M.H. 845-862. (with M.E. Chaftez and H.T. Blane)

1971a How People Perceive Technology. In Program on Technology and Society, Sixth Annual Report 21-24. Cambridge: Harvard University Press. (with D. Armor)

1971b A Cross-cultural Study of Trance. In Proceedings of the American Psychological Association.

1972 An Alcoholism Training Program from the Participants' Point of View. American Journal of Orthopsychiatry 42:318-319.

1976 Alcohol Consumption in Bars: Validation of self-reports against Observed Behavior. Drinking and Drug Practices Surveyor (11):13-15.

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Publications (cont'd.):

1978 Statement on Smoking Legislation. Congressional Record. September 7

1979 Pre-Columbian Tobaccos in the Pacific. Economic Botany 33(2):213-226. (with H.C. Fleming and J.M. Monahon)

1979a Drinking Patterns Among Single, Married, Divorced and Separated Women. Alcoholism: Clinical and Experimental Research III(2):179. (with Mary Christine Hoben)

1979b The Anti-Smoker: Searching for the Past. The Tobacco Observer 4(5):8-9.

1981 Social Function as a Component of Market Value. In Analysis of Consumer Policy. Wharton Applied Research Center, University of Pennsylvania.

Papers Read at Meetings and Invited Lectures:

1956 Report on the Earliest Cultural Affiliation with Amaranth Seeds in North America. American Association for the Advancement of Science. December. (with G. Agogino)

1958 Some Social and Clinical Factors Related to the Duration of Hospitalization. New York State Inter-Hospital Conference. April. (with R. Hardt)

1958a Social Class and Schizophrenia. New York State Sociological Society. May. (with R. Hardt)

1959 Recent History of the Extended Family in India- Part II. American Anthropological Association. October. (with H. Orenstein)

1959a Recent History of the Extended Family in India- Part I. Southwest Social Science Association. October. (with H. Orenstein)

1966 Diagnosis and Treatment in an East African Tribe. Brockton V.A. Hospital, 12th Annual Lecture Series. February.

1967 Some Ethnographic Observations on Drinking. Massachusetts General Hospital, Seminar on Alcoholism. February.

1967a Effects of Alcohol on Behavior. Brockton V.A. Hospital, 13th Annual Lecture Series. May.

1967b Diagnosis of Disease and Healing Categories in East Africa. Court St. Regional V.A. Hospital. July.

1967c Transcultural Psychiatry. Taunton State Hospital, Psychiatric Lecture Series. October.

1969 Human Side of the Boston Rehabilitation Project. Boston University, Institute of Innovative Techniques in Housing. January.

1969a Conflict and Conflict Resolution in a Roxbury Housing Program. Boston University School of Medicine, Scientific Colloquim. Sept.

1971 A Cross-Cultural Study of Trance. American Psychological Association, Annual Meeting. April.

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Papers Read at Meetings and Invited Lectures (cont'd.):

1972	Evaluation of an Alcoholism Training Program. American Orthopsychiatric Association, Annual Meeting. April.
1973	Drugs and the Counter-culture: An Anthropological Approach. University of Rhode Island, Police Training Institute. June.
1974	An Ethnographic Approach to the Streets, Drugs and Drug Treatment. Society for Applied Anthropology. Boston. March.
1976	An Ethnographic Study of Bar Behavior. American Psychological Association, Annual Meeting. September.
1978	Testimony before the Tobacco Sub-committee of the House Committee on Agriculture. Washington. September.
1979	Testimony before the Fuel Use Act Panel of the Department of Energy. Boston. February. (with Marcella Mazzarelli)
1979a	Drinking Patterns Among Single, Married, Divorced and Separated Women. National Council on Alcoholism Forum. March.
1980	The Social Role and Acceptability of Tobacco Use. R. J. Reynolds, Speakers Series. October.
1981	Social Function as a Component of Market Value. Conference on Consumer Policy, Wharton Applied Research Center. Philadelphia. May.

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STATEMENT OF EDWIN R. FISHER, M.D.

I am Edwin R. Fisher of Pittsburgh, Pa. I am currently professor of pathology at the University of Pittsburgh School of Medicine and director of laboratories at the Shadyside Hospital in Pittsburgh, Pa.

In addition, I am a consultant in pathology at the Veterans' Administration Hospital in Pittsburgh and the Brownsville General Hospital of Brownsville, Pa. I am a 1947 graduate of the University of Pittsburgh School of Medicine. I received postgraduate training at the Cleveland Clinic, Cleveland, Ohio, and the National Institutes of Health, Bethesda, Maryland.

I was certified by the American Board of Pathology in both anatomic and clinical pathology in 1952. I am a member of the honorary medical society Alpha Omega Alpha and Sigma Xi, the honorary society for scientific advancement.

I was the recipient of the Parke-Davis Award in Experimental Pathology in 1963 and the Man of the Year in Medicine in the city of Pittsburgh in 1966.

I am a member of many scientific societies, including the American Association of Cancer Research and the American Society for the Study of Arteriosclerosis.

I am the author of [illegible] scientific publications in American and international journals and textbooks.

I have served on the editorial boards of the journal Cancer and of the American Journal of Clinical Pathology as well as the Board of Scientific Directors of Ellis Fischel Cancer Hospital, Columbia, Mo., and the Board of Reviewers for the American Society of Atherosclerosis and am the project pathologist for the National Surgical Adjuvant, Breast and Colon Projects of the National Cancer Institute.

I should first like to direct my remarks to personally conducted experiments concerning the possible atherogenic effects of nicotine per se. Atherogenesis is the process which results in the disease which we commonly regard as hardening of the arteries, technically called atherosclerosis.

We have also conducted such experiments with actual cigarette smoke which, of course, includes the relationship of carbon monoxide to the atherosclerotic process. Reprints describing these experiments are being submitted as part of this statement.

In these experiments it was clearly demonstrated in the rabbit that realistic doses of either nicotine or cigarette smoke failed to initiate, exacerbate, or otherwise influence the atherogenic process in that species.

You will notice that I used the designation "realistic doses". I think that is very important. There have been some studies which have exhibited minor or questionable changes with the use of an equivalent dose of 600 or more cigarettes a day in man. This is such a large number that I think man would find it difficult to find the time to smoke them.

Another point which I would like to emphasize is this. One could justly say you found nothing in the rabbit but can the experience in the rabbit apply to man? What the scientific experiment in the laboratory can do is indicate where we should look and perhaps what we should look for in the clinical setting.

With those two points in mind, I should like to continue. It might be well to emphasize that there is no pharmacologic or other study of any scientific validity or acceptability to me that indicates that nicotine adversely affects coronary blood flow. Indeed, most of the studies reveal that this agent actually accentuates and enhances coronary blood flow. Accounts relating adverse effects of cigarette smoke on angina patients, that is, persons who suffer chest pain, probably as a result of arteriosclerotic heart disease, should not be interpreted as indicating that cigarette smoke is etiologically related to the arteriosclerotic process. Of course there are many things which a person with angina should not be doing. The issue is whether smoking causes the condition which manifests itself as angina. The answer is it hasn't been demonstrated.

Further it should be noted that the studies which purport to show that smoking already affects persons with angina involved very few patients -- less than 10. I submit to you that is not a valid scientific sample upon which to base any conclusions.

I have reviewed the scientific literature on the subject of coronary heart disease and smoking and find other studies which support my experimental work. For example, studies of workers exposed to carbon monoxide over long periods of time find that the exposed workers do not have an increased incidence of atherosclerosis compared to the general population.

Other studies raise serious questions about the smoking causation hypothesis. For example, studies of twins in Sweden are very illuminating on this subject. When the researchers looked at monozygotic twins (i.e., identical twins), they found that there was no increased rate of coronary heart disease in the smoking twin as compared to the nonsmoking twin. This led the researchers to conclude that their studies "...can be interpreted as showing that both the development of IHD [ischaemic or coronary heart disease] and death from it are under a relatively strong genetic influence."

Likewise, a 1979 study of cardiovascular disease in Switzerland found that while Swiss women have increased their smoking over the last quarter century, their rate of cardiovascular disease has declined significantly during the same period.

Thus, my own experimental work and review of the scientific literature leads me to the conclusion that cigarette smoking has not been scientifically established to be a cause of atherosclerosis.

CURRICULUM VITAE

EDWIN R. FISHER, M.D.

Date of Birth: 9/2/23

Education

University of Pittsburgh - B.S., Summa cum Laude, 1945.
University of Pittsburgh - School of Medicine, M.D., 1947.

Postgraduate Training & Hospital Appointments

Rotating Internship, Mercy Hospital, Pittsburgh, Pa. 1947-1948.
Residency in Pathology, Mercy Hospital, Pittsburgh, Pa. 1948-1949.
Residency in Pathology, Cleveland Clinic, Cleveland, Ohio. 1949-1951.
Assistant Pathologist & Staff, Cleveland Clinic, Cleveland, Ohio. 1951-1952.
Dept. of Pathology & Histochemistry, Natl. Inst. Health, Bethesda, Md. 1952-1954.
Associate Pathologist & Staff, Cleveland Clinic, Cleveland, Ohio. 1954.
Chief, Laboratory Service, Veterans Adm Hosp., Pittsburgh, Pa. 15240. 1955-1970.
Assoc. Chief of Staff, Veterans Adm Hosp., Pittsburgh, Pa. 15240. 1967-1970.
Director of Laboratories, Shadyside Hospital, Pittsburgh, Pa. 1970-
Consultant in Pathology, Veterans Adm. Hospital, Pittsburgh, Pa. 15240. 1970-

Teaching Appointments

Teaching Fellow, Dept. of Pathology, University of Pittsburgh. 1948-1949.
Associate Professor of Pathology, University of Pittsburgh. 1955-1958.
Professor of Pathology, University of Pittsburgh. 1958-

Certifications

Certified, American Board of Pathology in Pathologic Anatomy and Clinical Pathology, 1952.

Honorary Fraternities

A.O.A.
Sigma Xi

Awards

Senior Research Fellow, U.S.P.H.S. 1957-1961.
Career Research Award, U.S.P.H.S. 1962-1965.
Parke-Davis Award in Experimental Pathology, 1963.
Man of the Year in Medicine, Junior Chamber of Commerce of Pittsburgh, 1956.
Varsity Letterman of Distinction, Varsity Letter Club, University of Pittsburgh, 1971.

Societies

Am. Assn. of Pathologists & Bacteriologists.
American Society of Clinical Pathologists.
Society for Experimental Medicine & Biology.
Am. Assn. for Advancement of Science.
Am. Assn. of University Professors.
Assn. of Univ. Pathologists (Pluto Club).
Am. Society for Study of Arteriosclerosis.

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CURRICULUM VITAE (Continued)

Edwin R. Fisher, M.D.

Electron Microscope Society of America
American Society of Dermatopathology
Penna. Assn. of Clinical Pathologists
Am. Society of Nephrology
College of American Pathologists
International Academy of Pathology
Society for Experimental Pathology
American Rheumatism Association
Histochemistry Society
American Assn. for Cancer Research
American Society of Cell Biology
New York Academy of Science
American Therapeutic Society
Pan American Medical Society
American Thoracic Society
Transplantation Society
Arthur Purdy Stout Society of Surgical Pathologists
Robert B. Turnbull Surgical Society

Editorial Positions

Editorial Advisory Board, Cancer Metastasis Reviews
Consulting Editor, Year Book of Cancer
Board of Editors, European Journal of Cancer
Board of Editors, Americal Journal of Clinical PAthology
Board of Editors, Cancer 1973-1976
Board of Editors, Ultrastructural Pathology
Board of Editors, Breast Cancer Research and Treatment

Committees

Union International Contra Center, 1968-1971
Consultant, Committee on Nomenclature and Classification of Disease,
  College of American Pathologists, 1958-1962
Chief Pathologist, National Surcigal Adjuvant Breast Project, Cancer Institute, 1971-
Consultant Reviewer, American Heart Association, 1973-
Board of Scientific Advisors, Cancer Research Center, Ellis Fischel State Cancer Hospital,
  Columbia Missouri, 1975-
Referee, Faculty Scholar Award Program, Josiah Macy Jr. Foundation, 1978-

Publications

480 as of July 1, 1981.

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1. * Successful management of infantile diarrhea in a general hospital. Am. J. Dig.Dis., 17:334-337, 1950.
2. * Polyarteritis nodosa involving the female genital tract. Am. J. Ob. & Gyn. 60:445-447, 1950.
3. Observations on the eosinophil count in man: a proposed test of adrenal cortical function. Am. J. Med. Sci. 221:121-132, 1951.
4. Eosinophilic granuloma of [illegible]. J. Thoracic Surg. 21:24-29, 1951.
5. * Hamartoma of the pericardium: Lymphatic type. Clev. Clin. Quart. 18:12-17, 1951.
6. * Primary linitis plastica carcinoma of the colon. Clev. Clin. Quart. 18:210-217, 1951.
7. * Nodular goiter and carcinoma of the thyroid. Am. J. Surg. 82:202-208, 1951.
8. * Bleeding duodenal ulcer associated with cerebellar tumor in childhood. Gastroenterology 18:626-631, 1951.
9. * Intracranial ependymoma: Clinicopathologic observations. Clev. Clin, Quart. 18:260-270, 1951.
10. * Nonchromaffin paraganglioma of the orbit. Cancer 5:521-524, 1952.
11. * Histologic and distribution studies of radioactive gold in the guinea pig. Am. J. Med. Sci. 223:502-511, 1952.
12. Arrhenoblastoma of the ovary. Ann. Surg. 135:543-548, 1952.
13. * Coarctation of the abdominal aorta with resultant hypertension. Arch. Int. Med. 89:943-950, 1952.
14. * Linitis plastica carcinoma of the stomach with extensive metastases simulating a colonic lesion. Gastroenterology 20:303-308, 1952.
15. * Amyloid tumor of the urinary bladder. J.A.M.A. 50:96-98, 1952.
16. * The differentiation of invasive and non-invasive carcinoma arising in intestinal polyps. Clev. Clin. Quart. 19:62-67, 1952.
17. * Malignant polyps of the rectum and sigmoid: Therapy based on pathologic considerations. S.G. & O. 94:619-623, 1952.
18. Malignant polyp of the rectum: Follow-up observations on 27 patients. Post Graduate Med. 12:303-307, 1952.
19. * Hemangiopericytoma: Special histologic and tissue culture studies. Am. J. Path. 28:653-661, 1952.
20. * Thymic neoplasms. J. of Thoracic Surg. 24:58-69, 1952.
21. Pathologic changes following bilateral nephrectomy in dogs and rats. J. of Lab. Invest. 1:351-363, 1952.
22. * Multiple mesothelial cysts of the peritoneum. Am. J. of Surg. 84:328-330, 1952.
23. The relation between benign ulcer and carcinoma of the stomach. Gastroenterology 22:103-111, 1952.
24. The nephrotoxic action of spermine. Proc. Soc. Exper. Biol. & Med. 80:452-454, 1952.
25. Simultaneous occurrence of thyroiditis and papillary carcinoma of the thyroid. Cancer 6:57-62, 1953.
26. * Diffuse papillomatous polyps (villous tumors) of the colon: Pathologic and clinical observations. Am. J. Surg. 85:146-151, 1953.
27. * The destruction of cytoplasmic basophilia with mineral acids. Stain Tech. 28:9-12, 1953.

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Bibliography - Edwin R. Fisher, M.D.

28. * The pathology of a pneumotropic virus latent in C-3-H mice carrying the Bittner milk factor. Arch. Path. 55:14-19, 1953.
29. A naturally occurring tumor of gray squirrels related to the Shope rabbit fibroma. Proc. Soc. Exp. Bio. & Med. 82:298-301, 1953.
30. * Observations on thyroid colloid. Arch Path. 56:275-285, 1953.
31. Metachromatic basophilia of keratin after oxidation cleavage of disulfide bonds. J. Histochem. & Chtochem. 2:95-102, 1954.
32. * Nature and staining reactions of the fibroma cell inclusions of the Shope fibroma of rabbits. J. Nat'l. Cancer Institute 14:355-365, 1953.
33. Further observations on the role of bile in the pathogenesis of pancreatitis. Surg. Forum, Vol. 4:406-412, 1953.
34. The pathogenesis of fibroma in cottontail rabbits. Am. J. Hygiene 59: 104-112, 1954.
35. * Glomerular lipoidosis in dog kidney. Am. J. Vet. Res. 15:285-286, 1954.
36. * The effect of methylation on basophilia. J. Histochem. & Cytochem. 2:81-87, 1954.
37. * The pathology and pathogenesis of spermine induced renal disease. Arch. Path. 57:244-254, 1954.
38. Alkaline phosphatase and periodic acid-Schiff reaction in the proximal tubule of the vertebrate kidney: A study in segmental differentiation. Anat. Rec. 120:1-21, 1954.
39. Nephrotoxic action of spermine and related amines. Proc. XIX Internat. Physiol. Congress. 714-715, 1953.
40. The effect of increased hepatic blood flow upon liver regeneration. Arch. Surg. 69:263-272, 1954.
41. * Cytoplasmic liver cell inclusions following arterialization in the dog: Their nature, significance and relation to other experimentally induced cytoplasmic inclusions. Am. J. Path. 30:987-1003, 1954.
42. * The occurrence of cytoplasmic liver cell inclusions following arterialization in the dog. Fed. Proc. 13:428-429, 1954.
43. Liver regeneration following complete arterialization and increase in hepatic blood flow. Fed. Proc. 13:44, 1954.
44. * The differentiation of the Reed-Sternberg cell and megakaryocyte with special reference to the periodic acid-Schiff reaction. Lab. Invves. 3: 261-269, 1954.
45. * "Pseudomucinous" cytadenoma - A misnomer? Obst & Gyn. 4:616-621, 1954.
46. * Hepatic lesions of acute hemorrhagic pancreatitis: Their nature and pathogenesis. Surg. 37:213-219, 1955.
47. * The practical value of histopathological classification of gastric carcinoma: an appraisal based on 100 consecutive cases. Cancer 8:389-395, 1955.
48. * Whipple's Disease: Report of case apparently cured and discussion of the histochemical features. Clev. Clin. Quart. 21:213-221, 1954.
49. * Combined Gomori methods for the demonstration of pancreatic alpha and beta cells. Am. J. Clin. Path. 24:1433-1434, 1954.
50. * Satisfactory slide carriers for Stender and Coplin containers. Am. J. Clin. Path. 24:1435-1436, 1954.
51. * The cytologic demonstration and significance of tumor cells in the mesenteric venous blood in patients with colorectal carcinoma. S.G. & O. 100:102-108, 1955.
52. * Ovarian cystomas: Clinicopathologic observations. Cancer 8:437-445, 1955.
53. Effects of renin in rats treated with methylandrostenediol. Endocrin. 56:541-546, 1955.

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55. * Thrombotic thrombocytopenic purpura: Report of a case with discussion of its tinctorial features. Am. J. Clin. Path. 25:620-628, 1955.
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57. The sterilization and storage of lyophilized blood vessels. Ann. Surg. 143:73-80, 1956.
58. * Nor-epinephrine cells of the adrenal medulla following hypothermia and unilateral adrenalectomy in the rat. Proc.Soc.Exper. Biol. & Med. 89: 140-142, 1955.
59. Further experimental observations on animals with arterialized livers. Surg. 38:181-193, 1955.
60. Renal lesions of sulfonamide type following administration of acetazolramion (Diamox). J.A.M.A. 160-204-206, 1956.
61. * Darier's disease of the larynx. A.M.A. Otolaryng. 62:438-441, 1955.
62. * Neuroblastomas of the nasal fossa. A.M.A. Arch. Path. 60:435-439, 1955.
63. * The nature of the periductal stroma in gynecomastia. Lab. Invest. 5:267-275, 1956.
64. The hepatotoxic effect of nitrogen mustard following direct intraportal injections. Cancer 9:144-147, 1956.
65. * Histochemical observations on an alveolar soft part sarcoma with reference to histogenesis. AM. J. Path. 32:721, 1956.
66. Fate of autogenous skin tubes as aortic grafts. S.G. & O 102:309-314, 1956.
67. Experimental reconstruction of the aortic bifurcation. Surg. 39:940-949, 1956.
68. * The effect of induced arteriosclerosis on fresh lyophilized aortic homografts in the rabbit. Surg. 40:529-532, 1956.
69. * Malignant blue nevus. A.M.A. Arch Dermatol. 74:227-231, 1956.
70. Sex differences in the spread of colorectal and vesicle carcinomas. Ann. Surg. 143:284-286, 1956.
71. * Clot formation in the common duct an unusual complication of primary hepatic carcinoma. A.M.A. Arch Surg. 73:261-265, 1956.
72. The effect of hypothermia upon induced bacteremia. Fed. Proc. 15:514, 1956.
73. * The nature of diffuse intra-arteriolar occlusion in patients with carcinoma. AM. J. Path. 32:1185-1203, 1956.
74. * Does the aortic homograft develop arteriosclerosis? An experimental study. Clev. Clin. Quart. 23:159-160, 1956.
75. * Parathyroid cysts. Cancer 10:57-62, 1957.
76. Postnatal change in the alkaline phosphatase activity and the periodic acid-Schiff reaction of the mouse kidney. Q.J. Mic. Sci. 97:187-195, 1956.
77. * Extramammary Paget's disease. Amer. J. Surg. 94:493-498, 1957.
78. Observations concerning vacuolation and deposition of glycogen in nuclei of hepatic cells. J. Lab. Invest. 6:324-333, 1957.
79. The effect of hypothermia upon induced bacteremia. Proc. Soc. Exper. Biol. & Med. 93:510-512, 1956.
80. * Polyarteritis nodosa associated with rheumatoid arthritis. AM. J. Clin. Path. 27:191-204, 1957.
81. Paper electrophoresis of serum: methods and application. Penn. Med. J. 60:972-976, 1957.
82. * Some practical applications of histochemistry. Penn. Med. J. 60:1467-1472, 1957.

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84. * Stressor effect of hypothermia in the rat. Am. J. Physiol. 188:470-472, 1957.
85. * Multiple endocrine tumors and peptic ulcer. Gastroenterology 32:1075-1094, 1957.
86. Bronchiolar emphysema (so-called muscular cirrhosis of the lungs). Amer. J. Path. 33:1137-1162, 1957.
87. A histochemical basis for changes in renal tubular function in young mice. Extrait des Comptes Rendus le l'Association des Anatomistes (XLII) Reunion Paris, 25-30 jiullet, 1955).
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89. * Alteration of renal succinic dehydrogenase in nephrotoxic serum nephrosis. Fed. Prox. 16:356, 1957.
90. * Pathologic and histochemical observations in experimental hypothermia. Arch. Surg. 75:817-827, 1957.
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93. Forced horizontal conjugate gaze in man. J. Comp. Neurol. 110:257-269, 1959.
94. Hemiballismus secondary to metastatic carcinoma of gallbladder. Neurol. 7:873-874, 1957.
95. Epidermal cyst in bone. Cancer 11:643-648, 1958.
96. * Post inflammatory tumor (xanthoma) of lung. Dis. of Chest 36:43-48, 1959.
97. Observations concerning bacterial defense mechanisms during hypothermia. Surg. 43:807-814, 1958.
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106. * Pathogenesis of pancreatitis. Am. Practitioner and Digest of Treatment 9:1253-1255, 1958.
107. * Cytomegalic inclusion disease in the adult. New Eng. J. Med. 258. 1036-1040, 1958.
108. Observations on the treatment of brain abscess by direct instillation of newer antibiotics. Univ. of Mich. Med. Bull. 24:61-65, 1958.

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111. * Pathologic findings in a case of panhypopituitarism and diabetes insipidus: comments relative to hypothalamic, pituitary and "end-organ" interrelationships. A.M.A. Arch. Path. 66:282-293, 1958.

112. * Perianal rhabdomyosarcoma. A.M.A. Arch. Surg. 77:230-234, 1958.

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114. * Myocarditis with endocardial elastomyofibrosis (EEMF). Am. Heart J. 56:537-552, 1958.

115. * Observations concerning the pathogenesis of endocardial thickening in the adult heart. Am. Heart J. 56:553-561, 1958.

116. Renal lesions of aminonucleoside nephrosis as revealed by electron microscopy. Lab. Investigation 8:371-385, 1959.

117. * Laboratory and clinical differentiation of chronic glomerulonephritis and nephrosclerosis. Am. J. Med. Sci. 236:551-566, 1958.

118. * Ossifying metastatic carcinoma: Report of a case with comments relative to histogenesis of ectopic ossification. Cancer. 12:257-262, 1959.

119. The favorable effect of pyridoxine deficiency on skin homograft survival. Surgery. 44:49-167, 1958.

120. Thrombotic thrombocytopenic purpura (TTP). A cause of massive gastro-intestinal hemorrhage. Gastroenterology 36:132, 1959.

121. * Differentiation of neoplastic lesions characterized by large, vacuolated intraepidermal (Pagetoid) cells. A.M.A. Arch. Path. 67:140-146, 1959.

122. Reticulum cell sarcoma of brain (microglioma) A.M.A. Arch. Neurol. & Psychiat. 81:591-598, 1959.

123. * Experimental studies of factors influencing hepatic metastases I. Effect of number of tumor cells injected and time of growth. Cancer.12:926-928, 1959.

124. Experimental studies of factors influencing hepatic metastases II. Effect of partial hepatectomy. Cancer. 12:927-932, 1959.

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STATEMENT

OF

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I am H. Russell Fisher, of Glendale, California, a doctor of medicine. I am Emeritus Professor of Pathology at the University of Southern California, and former Director of the Department of Pathology at the Memorial Hospital of Glendale — where I am now on staff as a consultant. Until last year, I was Director of the Department of Pathology at the Santa Fe Hospital in Los Angeles, where I am still on the active staff as a research associate. I am a member and past officer of medical and scientific societies, including the National Research Council Committee on the International Council of Societies of Pathology. My special investigative interest has been in the field of carcinoma in situ. My full Curriculum Vitae is submitted with this statement.

As a medical scientist, I am concerned with the legislative proposal now termed the "Comprehensive Smoking Prevention Act of 1982." It would be inappropriate to adopt it on several grounds. Its basis is contrary to a significant body of scientific knowledge about the putative role of cigarette smoking in the causation of disease. It misuses the scientific definition of "cause" as that term relates to disease. Its justification rests in part upon statements that have not been found to be valid. Moreover, the bill creates an unnecessary additional layer of bureaucracy, duplicating many functions already in place and available in various agencies in the government; this may well hamper solid scientific research. The National Institutes of Health and the National Cancer Institute are already expert in reviewing grant applications.

In 1969, I submitted a statement to Congress in which I said that I, like many of my fellow scientists, was puzzled over the cause of lung cancer, and that, on the basis of the evidence then available, no one knew the cause of cancer of the lung. Contrary to the proposed "findings" in the bill, we still do not know the cause or causes of lung cancer; nor do we know what role, if any, smoking plays in the causation of this disease.

Most of the claims made against cigarettes are based on statistical coincidence, or statistical association, from which conclusions can be drawn only if all the facts of nature are known, which they are not. This statistical coincidence has given rise to an emotional tide, seized upon by various groups and agencies, in the hope to fill a void in our efforts to achieve a perfect health.

Like most pathologists, I am not expert in the use of mathematical systems or statistics, but there are a few basic rules that are easy to apply. For example, each population group used in the statistical analysis must have uniformity and homogeneity. Data must be reliable and comparable and, most importantly, the reference assumptions must have an existence in fact.

Most, if not all, of the epidemiological studies reporting a statistical correlation between cigarette smoking and lung cancer do not meet these simple criteria. For example, they rely primarily on death certificates for the diagnosis of lung cancer even though studies have shown great unreliability in the death certificate specification of this disease. This is primarily due to confusion between cancers that arise in various parts of the body and spread to the lungs - metastatic cancer - and cancers that start in the lungs - primary cancer.

Sommers, participating on a committee of the New York Academy of Medicine to evaluate the accuracy of death certificate diagnosis, found them to be correct in less than 50% of the cases. And Rosenblatt, in a series of studies on the diagnosis of patients dying in the Doctors Hospital of New York during the last decade, found that the clinical diagnosis of primary carcinoma of the lung was confirmed by autopsy examination in only 45% of the cases.

In 1979 Saxen reported on the difficulty of achieving accurate death certificate diagnoses, working with data from Finland, a small country with a stable population. Reviewing 162 deaths attributed to respiratory cancer in a 2 year period, he found, on microscopic tissue examination, that 35 cases (22%) had to be ruled out as not having primary lung cancer.

One thing that has added to the difficulty of this problem is the controversy about the various histological types of lung cancer and their relationship to causation. Some years ago Kreyberg, a famous lung pathologist of Oslo, classified cancer of the lung into two major types or groups. One group was primarily made up of squamous cell carcinoma and the other adenocarcinoma. In squamous cell carcinoma the cells grow as flat plates, like the cells of our skin. In adenocarcioma the cells are cylindrical and grow in rings, like glands.

Kreyberg proposed the idea that the reported rise in the incidence of lung cancer was due to a rise in incidence of squamous cell carcinoma and that this kind of lung cancer was caused by smoking and other environmental factors. According to Kreyberg, adenocarcinoma was unrelated to environmental factors. Somehow this idea that smoking caused some types of lung cancers but not others was accepted by many, even though they could not agree with Kreyberg on his standards for classification.

Because many investigators associate cigarette smoking with squamous cell carcinoma, there has been continuing interest in the trends of squamous cell carcinoma versus adenocarcinoma. The study by Vincent et al., from Roswell Park showed that since 1963 squamous cell carcinoma has dropped and adenocarcinoma has increased. In contrast, a Mayo Clinic study showed a progressive increase in squamous cell carcinoma. Such differences are probably due to a lack of uniformity in the standards of microscopic criteria, and statistical selection emphasize the need for homogeniety in elements subjected to statistical comparison.

The controversy about tumor types is particularly pertinent to the issue of lung cancer in women. It is claimed that the incidence of female lung cancer is rising due to their increase in smoking since World War II. However, studies indicate that the purported rise is primarily due to an increase in adenocarcinoma, the type not generally associated with smoking. For example, Dr. Ioachim from Dr. Sommers' pathology department at the Lenox Hill Hospital (N.Y.), finds that adenocarcinoma makes up about half of the cancers in females.

That the smoking and lung cancer controversy continues is amply demonstrated by the recent study which appeared in the April 1979 issue of the Journal of the National Cancer Institute. This study challenged the dogma that smoking is the major cause of lung cancer. Using data from a number of broad studies of lung cancer, including the American Cancer Society study population, Enstrom showed that there has been a dramatic rise in lung cancer mortality rate among persons who have never smoked. In white females who have never smoked, the increase in lung cancer in the last 4 decades has paralleled the increase in those who smoke cigarettes. This negates the claim of the American Cancer Society that the increase in lung cancer in females over that period has been due to cigarette smoking. Enstrom noted that "Many factors other than personal cigarette smoking have not been exmained in great detail in their relationship to lung cancer."

We just do not know the cause of lung cancer despite a mountainous accumulation of research on the subject. Scientists have not produced the kind of lung cancer associated with smoking — squamous cell carcinoma — in animals exposed to cigarette smoke. Experimental work simply does not support the idea that lung cancer is caused by cigarettes and scientists are looking elsewhere for the cause of cancer, into genetic, environmental, and viral possibilities. The federal government, through the National Cancer Institute is now funding research into these possibilities.

There is no need to create a new office of smoking and health to collect the results of research on the effects of smoking, or to facilitate such research. This could be done adequately by capacities already in place in the Department of Health and Human Services by assignment to a single qualified individual qualified in bibliographic compilation and by a directive from the Head to those organizations already expert in reviewing grant applications, such as the National Institute of Health and the National Cancer Institute. A new bureau just isn't needed nor is it necessary or appropriate to have a new complex committee on educating the public until there is a firmer foundation of scientific knowledge of what is to be taught, what reliable information is to be disseminated.

Knowledge from medical and scientific research must be interpreted with great care and with an understanding of the great variability of the biological processes. Any action having the force of the Congress of the United States should be based on fact, not conjecture. Experience has shown that social legislation not based on valid information has an unhappy history.

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  Member, Board of Governors

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California Medical Association

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California Society of Pathologists:
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Los Angeles Society of Pathologists:
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STATEMENT OF JEAN D. GIBBONS

My name is Jean Dickinson Gibbons. My current position is Professor of Statistics and Chairman of the Applied Statistics Program at the Graduate School of the University of Alabama. I am currently a Fellow of both the American Statistical Association and the International Statistical Institute and a member of the Committee on National Statistics of the National Academy of Sciences.

I received the bachelor's and master's degrees in mathematics from Duke University and the Ph.D. degree in statistics from Virginia Polytechnic Institute and State University. My previous faculty appointments were at the University of Pennsylvania and the University of Cincinnati. I was a senior Fulbright-Hays scholar at the Indian Statistical Institute in 1973.

I was Associate Editor of The American Statistician for eight years, currently act as editorial collaborator on many statistical journals, including The Journal of the American Statistical Association, Biometrics, and Technometrics, and serve as a reviewer for grant proposals to the National Science Foundation. I am a member of several professional societies and have served two terms on the Board of Directors of the American Statistical Association.

My publications include four scholarly books on statistics and over 30 articles in refereed professional and learned journals in my field. I was named Outstanding Scholar in 1981 and Board of Visitors Research Professor in 1974 at the University of Alabama. My current curriculum vita is attached to this statement.

In February of 1978, I was asked to review the reported statistical studies that formed the basis for the Food and Drug Administration's (FDA) decision to include a boxed warning in the patient and physician labeling on oral contraceptive (OC) products which states that cigarette smoking increases the risk of serious cardiovascular side effects for OC users. In October of 1978, at the request of Representative L. H. Fountain, I testified as an expert witness at a House Subcommittee Hearing on the "Quality of Scientific Evidence in FDA Regulatory Decisions (The Adoption of an Antismoking Warning in Oral Contraceptive Pill Labeling)." My conclusion at that time was that the statistical evidence published in the literature about the interactive effects of smoking and oral contraceptives on risk of cardiovascular disease is quite weak because the sample sizes in most studies are extremely small, the results may be subject to significant sampling errors, and the results in some studies are based on convenient but unfounded assumptions. The authors of these papers in many cases pointed out these limitations of their data and deficiencies in their analyses. At that time I suggested that the FDA should run a controlled experiment to obtain sufficient and relevant data on factors such as length of time of OC use; number of years and amount of smoking; genetic, environmental, and psychological characteristics; among others. My prepared testimony and the discussion following at that hearing are a part of the written record.

Bill H.R. 4957 contains a finding that states "(5) women who take birth control pills and smoke are more likely to suffer a heart attack or stroke than women who don't smoke". This finding is similar to the wording on the patient insert warning on boxes of OC which was at issue in my previous testimony. Therefore, when Representative Fountain again contacted me in February of this year on this matter, I examined and reviewed the statistical studies that have been published on this topic since my previous analysis. My current study included over 20 papers, in addition to the 13 papers examined for the previous testimony and the 3 additional papers that appeared in 1978 and were mentioned during the questioning at that hearing. A complete list of references for the papers appearing since 1978 that I have studied is attached to this statement.

This statement includes a brief analysis of each of the relevant studies published since 1978. I have looked carefully and objectively at the data, findings, and conclusions of the authors, and also performed some independent calculations to measure the statistical relationship between smoking and OC use in their data and to check their conclusions. I have critically analyzed those papers in which the authors claimed there is no (or a slight) increased risk of heart attack or stroke for women who smoke and use OC, as well as those papers where the authors claimed that there is a definite and increased risk of heart attack or stroke. The papers which I reviewed but have not included in this written analysis are, in my opinion, either not relevant to the finding stated in H.R. 4957, or do not add any new results of significance, or do not help clarify the situation.

My primary overall conclusion is that Congressional Finding (5) in Bill H.R. 4957 is at present groundless because the statistical evidence on which it is based is limited, weak, controversial, and subject to severe criticism by impartial experts. There are current studies in the literature that, while also subject to criticism, have come to the opposite conclusion and indeed claim that there is no interactive effect of OC use and smoking on the occurrence of heart attack or stroke in women. The evidence for either conclusion is limited and weak and subject to serious scientific criticism. In my professional judgment, I believe that the Congress should not in good conscience find that "women who take birth control pills and smoke are more likely to suffer a heart attack or stroke than women who don't smoke" because the scientific evidence is inadequate. Most of the women who will be affected and influenced personally by this finding will not have the scientific background to form their own conclusions and will interpret the finding as truth, when, in fact, it is at best a questionable opinion that has been neither confirmed nor denied by the data in the reported studies. I again strongly urge the Congress to recommend that a controlled study be carried out with a good data base so that the issue can be addressed properly. More research is urgently needed before Congress can make a finding of such public importance.

In support of these general conclusions, I have attached a brief summary of my analyses of (I.) the group of reported studies that claim no increased significant risk of heart attack and/or stroke, and (II.) the group of reported studies that do claim an increased risk for women who smoke and use oral contraceptives. A complete list of references is attached. Additional details are given in the Appendices.

Analysis of Reported Studies

I. Studies which purport to find no (or slight) increased risk of heart attack and/or stroke for women who smoke and use OC.

A. Krueger et al. (1980) report a collaborative case-control study of death from myocardial infarction (MI) in women aged 15-44 for the period January 1974-June 1975 in regions representing the five largest metropolitan areas in the United States. Data on smoking habits and OC use were reported for 163 women who died of MI and 326 control women; these are reasonable sample sizes on which to base a conclusion and these data appear reasonably reliable.

A primary stated conclusion of the authors is "An interactive effect of OC use and smoking on risk of MI, as reported in recent studies in the U.S. of nonfatal MI, was not found . . ." (p. 672). My independent statistical analysis to verify their conclusion of no interactive effect, shown in Appendix A, is based simply on the reported numbers of smokers and OC users among the cases. My three conclusions from this independent analysis are as follows:

1. In the population of 163 women aged 15-44 who died from MI, the factors of cigarette use and OC use show no statistical association.

2. In the population of 44 women aged 15-44 who died from MI and had no predisposing conditions, the factors of cigarette use and OC use show no statistical association.

3. In the population of 119 women aged 15-44 who died from MI and had predisposing conditions, the factors of cigarette use and OC use show no statistical association.

Another primary stated conclusion of the authors is "Smoking and OC use together appeared to be no more of a risk factor for fatal MI than either smoking or OC use separately, compared to a reference group of nonsmokers and non-OC users" (p. 667). The authors based this conclusion on the odds ratios reported in Table 13, p. 666, and I have verified these odds ratios in Appendix A by independent calculations. The odds ratios and 95% confidence limits (from Table 13, p. 666) are as follows:

Nonsmokers, OC users	2.19	(0.60, 7.33)
Smokers, non-OC users	2.15	(1.38, 3.39)
Smokers, OC users	1.84	(0.81, 4.06)

The fact that the confidence interval for smokers and OC users includes 1.0 is statistical evidence that there is not necessarily any increased risk of MI for women who smoke and use OC over those who do neither; the same conclusion applies to women who do not smoke and do use OC. The confidence level for each of these statements is .95. The fact that the odds ratio for smokers and OC users is smaller than the odds ratios for each of the groups (Nonsmokers, OC users) and (Smokers, Non-OC users) in fact implies the opposite of a synergistic effect.

It is unfortunate that the paper does not give information on the joint characteristics of cigarette and OC use among the deceased controls so that the results could be compared for women who died from MI and women who died from other causes. It is also unfortunate that the data given on both cases and controls have no breakdown according to amount of smoking, years of OC use, and age category within the 15-44 years. Surely the duration of OC use, duration of smoking, amount of cigarettes smoked, and age are important factors to consider in determining whether a relationship exists.

B. Slone et al. (1981) report on a case-control study of the rate of nonfatal MI with respect to the characteristics of duration of current and past OC use, three subcategories of age group within 25-44 years, and four categories of smoking status. The only data on rate-ratios provided in the paper that concern smoking status and OC use are as follows (from Table 7, p. 423):

Never Smoked	2.5
Ex-smokers	2.9
1-24 cigarettes/day	1.5
≥ 25 cigarettes/day	1.4

The authors conclude from these findings that "The rate-ratio estimates declined with increasing cigarette smoking, and the trend was statistically significant. This finding is at variance with previously published observations on current use of oral contraceptives" (p. 423). However, the authors warn of possible bias in this study and recommend that the data be interpreted with caution.

C. Maguire et al. (1979) extend the analysis of some previously reported data on a case-control study of four diagnostic categories of thrombosis (including MI). The primary conclusion relevant here is that their analysis indicates "no strong evidence of modification in the relative risk associated with oral contraceptive use by age or smoking for any of the thrombosis diagnoses considered. It is of interest, however, that in all groups except predisposed venous thrombosis the effect modifier coefficients were negative, suggesting a consistent pattern of decreasing estimated relative risk associated with pill use with both smoking and older age" (p. 193).

D. Belsey et al. (1979) use vital statistics from 21 countries on mortality from cardiovascular disease (CVD) for 1962-74, taking the pre-pill period as controls and post-pill period as OC users. (This is a re-examination of some data used by Beral for a 1976 publication in Lancet, but with an additional two years included.) These authors "find the conclusion of increasing mortality from cardiovascular disease associated with increased levels of pill use unsupported by the data" (p. 85). Their analyses fail to show a significant correlation between percent of women smoking and changes in CVD mortality. The authors do point out, however, the inadequacy of using vital statistics as opposed to a case-control study data base.

E. Jick et al. (1978c) report on a case-control study of nonfatal stroke in premenopausal women. They conclude "In our study, as in the report by the CGSS [Collaborative Group for the Study of Stroke in Young Women], cigarette smoking was only weakly associated with stroke in healthy young women" (p. 59). My independent analysis of their data, given in Appendix B, shows that cigarette smoking is not associated with stroke in this group of women. These authors also state that their "results indicate that oral contraceptives markedly increase the risk of stroke in healthy young women" (p. 59). My independent analysis of their data, also given in Appendix B, shows that there is indeed a positive association between OC use and incidence of stroke at the .001 level of significance. It should be pointed out that the sample sizes for this study are quite small, a total of 56 control and 14 case subjects.

F. Petitti et al. (1978b) use the Walnut Creek Data to study OC use, smoking, and other risk factors for venous thromboembolism and conclude "that OCs and smoking have independent effects in increasing the risk of the idiopathic form of the disease" (p. 484). Their conclusions are based on 17 cases without predisposing conditions grouped as follows:

Table: Number of Smokers and OC Users Among the 17 Cases (from Table 4, p. 483).

	Non-OC Users	OC Users	Totals
Nonsmokers	2	4	6
Smokers	6	5	11
Totals	8	9	17

$\chi^2_1 = 0.7 \quad .30 < P < .50$

Statistical Conclusion: Among women who have the disease and no predisposing conditions, the factors of cigarette use and OC use show no association.

The authors also give relative risk estimates for cases versus controls, and the estimates for smokers and OC users are smaller than the estimates for women with only one of these factors.

Table: Relative Risk Estimates for Cases vs. Controls (from Table 4, p. 483)

	Relative Risk	90% Confidence Limits
Among Smokers, OC users	4.7	(1.3-17.6)
Among Nonsmokers, OC users	12.8	(1.8-90.2)
Among OC Users, Smokers	2.3	(0.8- 7.1)
Among Non-OC Users, Smokers	7.6	(1.6-36.2)

Because the sample sizes for cases are so very small and the confidence intervals are so wide, these results are of questionable reliability.

II. Studies which do purport to find a definite and increased risk of heart attack and/or stroke for women who smoke and use OC.

A. Studies criticized in my previous testimony:

(1) Jain (1977) uses the data in Mann et al. (1975) consisting of 63 women under age 45 who had survived an MI and a control group. Among the cases, there were three nonsmokers and 13 smokers who were using OC at the onset of the MI episode. These numbers are too unbalanced and too small to justify any reliable conclusions about the interrelationship of smoking and OC use on MI. Jain concedes in his paper that these mortality data are based on small numbers and may be subject to significant sampling errors; this

caveat should not be ignored. Further, his analysis is based on two unwarranted but convenient assumptions which he justifies making ". . . because the relevant data . . . are not available" (p. 51). This is a non sequitur, and highly unscientific reasoning.

(ii) Beral (1977) uses the Royal College of General Practitioners (RCGP) data but the numbers are still very small, especially the deaths for nonsmokers. She concedes that "These estimates are based on small numbers and are necessarily approximate. Without more data it is not possible to examine the interrelationships of age, smoking, and duration of oral contraceptive use . . ." (p. 730).

(iii) Jick et al. (1978b) report data on 26 women with acute but nonfatal MI and 59 controls and give relative risk estimates for OC users. However, no MI subjects were nonsmokers who did not take oral contraceptives so relative risk estimates for OC users who smoke could not be obtained.

(iv) Petitti and Wingerd (1978) give relative risk factors for subarachnoid hemorrhage (SAH) for women who smoke and use OC. However, this analysis is based on extremely small numbers, a total of 11 women, which includes none who were nonsmokers and non-OC users, and only six who were smokers and OC users. Certainly this is insufficient data to use as a base for claiming an interrelationship.

B. Shapiro et al. (1979) report a study of 234 premenopausal women with MI and 1742 control premenopausal women. The authors give the following age-adjusted rate ratio estimates and 95% confidence intervals of MI for recent OC users (from Table V, p. 745):

Nonsmokers	4.5 (1.4-14.1)
Smoke 1-24/day	1.2 (0.3- 4.4)
Smoke $\geq$ 25/day	4.3 (2.2- 8.2)

The estimated rate ratio for heavy smokers is about the same as for non-smokers and the confidence interval for nonsmokers completely includes the confidence limits for heavy smokers. Further, the rate ratio estimate for moderate smokers is considerably smaller than either of those for nonsmokers and heavy smokers, and the confidence interval for moderate smokers includes the value 1.0, which shows no significantly greater risk.

The age-adjusted rate-ratio estimates given in Table VI (p. 746), on the other hand, give a very large rate-ratio estimate for women who are heavy smokers. The authors state that the Table VI results were derived from the data shown in Table V, but the results in Tables V and VI are so inconsistent that I do not see how they could have been obtained from the same data base. Further, the confidence intervals in Table VI are extremely wide for all categories of OC use and similarly for non-OC users who are heavy smokers. The confidence limits for moderate smokers who use OC include the value 1.0, a result that is consistent with the Table V results, but this again implies no risk. My independent calculations of rate ratios from the data in Table V without adjusting for age, as shown in Appendix C, are more consistent with Table V ratios than are those given by the authors in Table VI.

Perhaps more important here is the small frequency of women in each group. See Appendix C. These limited sample sizes alone could justify an argument that the results given by Shapiro et al. are not reliable.

C. Jick et al. (1978a) extend an earlier case-control study of nonfatal MI and its relation to OC use and smoking to include a total of 83 case and 154 control subjects. The authors claim a strong positive association between MI and OC use, and between MI and smoking. My independent analyses of their data, shown in Appendix D, confirm these conclusions. However, these authors also state "In both groups there is an extremely strong correlation between smoking and MI. Of the 83 case patients interviewed, 74 (89%) were current smokers. The corresponding figure for the 153 controls is 67 (44%)" (p. 2,549). As this statement shows, it is certainly true that the percentage of smokers is larger in the case group than in the control group, but this fact has nothing to do with correlation; in fact, the percentages have no relevance for the relationship between MI and smoking because the data bases were not random samples of cases and controls. This paper gives no data on the joint frequences of women wit respect to MI, OC use or smoking characteristics.

D. The NIH Report (1981) states "the Walnut Creek data also confirm that OC users who smoke or who are older than 30 years are at somewhat [emphasis added] greater risk of serious side effects, particularly circulatory disorders" (p. 1,071). However, no specific data or results are given to clarify what the degree of greater risk really is estimated to be.

E. Petitti et al. (1979) also use the Walnut Creek data. Table 1 (p. 1,152) shows the relative risks for MI as 2.9 for women who smoke and 2.8 for women who smoke and use OC (note this relative risk is lower), and the lower 90% confidence limit for women who smoke and use OC is 0.8, less than 1.0; these results for MI cases are based on a total of 26 observations, however. Other relevant results in this table are relevant risks for SAH of 5.7 for smokers and 21.9 for women who smoke and use OC, based on a total of 11 observations, and relative risks for other stroke as 4.8 for smokers and 2.0 for women who smoke and use OC (note this relative risk is lower than the relative risk for smokers), based on a total of 23 observations. Table 3 (p. 1,152) gives incidence rates for the combined types of cardiovascular disease for 3 women under age 45 who neither smoke nor use OC, and 8 women under age 45 who both smoke and use OC. All of the aforementioned results or relative risks are highly questionable because of the extremely small numbers of cases in each subgroup (especially that of women under age 45).

In spite of this severe limitation on reliability, the authors' conclusion is that "smoking and OC use appear to act synergistically to increase the risk of subarachnoid hemorrhage, hemorrhagic stroke, and MI" (p. 1,154). In my opinion, their data do not justify this conclusion at all.

F. Layde, Beral, and Kay (1981) use the RCGP data to study the relationship between smoking and OC use in regard to mortality from SAH and from various circulatory diseases. An independent analysis of their data in Tables IV and V (p. 543), shown in Appendix E, implies that the following statistical conclusions are appropriate: In the population of women aged 35-44, there is no association between OC use and smoking for those who died from SAH, nor for those who died from circulatory disease.

The sample sizes here are small (20 deaths from SAH and 65 deaths from circulatory disease). The authors use the 65 deaths from circulatory disease to compute the relative risk and excess risk estimates for Ever-users vs. Controls in Table V (p. 543), separately for each subgroup of age and smoking. The individual numbers of cases for each of these subgroups are extremely small (see Appendix E, Table 14). The authors do not use these actual numbers to compute relative risks and excess risks; rather they use mortality rates per 100,000 women-years and this leads to an impression of much larger sample sizes. Moreover, the authors state "The relative risk [of circulatory disease] for ever-users was greater among smokers than among non-smokers for each age group" (pp. 543-544). The authors fail to point out that the 95% confidence interval of relative risk for nonsmokers is considerably wider than that for smokers for women aged 35-44, and also for women 45 and over; and in fact the interval for nonsmokers includes the interval for smokers in each case, which implies no significant difference between relative risks for smokers and for nonsmokers.

C. Rosenberg et al. (1980) study the effect of OC use on nonfatal MI in the presence and absence of other predisposing risk factors including cigarette smoking. Their relative risk estimates and 95% confidence intervals for MI for women without other predisposing conditions are as follows (From Table 4, p. 63):

Normotensive nonsmokers	2.8	(1.0-7.8)
Normotensive smokers	1.1	(0.5-2.6)

These risk estimates are for current OC users relative to women who had never used OC.

The relative risk for normotensive smokers is smaller than that for normotensive nonsmokers, and the lower confidence limit is 0.5, which implies that the additional risk for smokers using OC is probably nonexistent. And yet the authors claim "The increase in risk attributable to the combined effect of current OC use, cigarette smoking and hypertension was considerably greater than what would be predicted from the sum of the separate effects of these factors" (p. 59).

Each of the risk estimates given in Table 4 (p. 63) is based on a very small number of cases, however. Only 7 cases were OC users and smokers without other predisposing conditions, and only 12 cases were neither smokers nor OC users. Further, the data base is married U.S. female registered nurses, which is hardly representative of all U.S. females.

Appendix A

Analysis of Data from Krueger et al. (1980)

Table 1. Number of Smokers and OC Users Among the 163 Cases (from Table 13, p. 666)

	Non OC Users	OC Users	Totals
Nonsmokers	42	6	48
Smokers	101	14	115
Totals	143	20	163

$\chi^2_1 = .0033$, $.90 < P < .95$

The appropriate statistical conclusion from Table 1 is that in the population of women aged 15–44 who died from MI, the factors of Cigarette Use and OC Use show no association.

Table 2. Number of Smokers and OC Users Among the 44 Cases Without Predisposing Conditions (from Table 14, p. 666)

	Non OC Users	OC Users	Totals
Nonsmokers	6	1	7
Smokers	31	6	37
Totals	37	7	44

$\chi^2_1 = .016$, $P = .90$

Table 3. Number of Smokers and OC Users Among the 119 Cases with Predisposing Conditions (from Table 14, p. 666)

	Non OC Users	OC Users	Totals
Nonsmokers	36	5	41
Smokers	70	8	78
Totals	106	13	119

$\chi^2_1 = .1038$, $.70 < P < .80$

The appropriate statistical conclusions from Tables 2 and 3 are that in the population of women aged 15-44 who died from MI, the factors of Cigarette Use and OC Use show no association, irrespective of whether there are predisposing medical conditions.

The odds ratios associated with OC use and smoking as given in Table 13, p. 666, are simple proportions of cases versus controls in each smoking category relative to the same proportion for nonsmokers, non-OC users, calculated as follows:

Nonsmokers, non-OC users	Reference Category	= 1.0
Nonsmokers, OC users	6/9 ÷ 42/138	= 2.19
Smokers, non-OC users	101/154 ÷ 42/138	= 2.15
Smokers, OC users	14/25 ÷ 42/138	= 1.84

Appendix B

Analysis of Data from Jick et al. (1978c)

Table 4. Number of Cases and Controls Who Smoke or Not

	Control	Stroke	Totals
Nonsmoker	33	7	40
Smoker	23	7	30
Totals	56	14	70

$\chi^2_1 = .36, \quad .50 < P < .70$

Statistical Conclusion: There is no association between incidence of nonfatal stroke and smoking in these women.

Table 5. Number of Cases and Controls Who Use OC or Not

	Control	Stroke	Totals
Non-OC User	49	3	52
OC User	7	11	18
Totals	56	14	70

$\chi^2_1 = 25.60, \quad P < .001$

Statistical Conclusion: There is a significant association between incidence of nonfatal stroke and OC use in these women.

Appendix C

Analysis of Data from Shapiro et al. (1979)

Table 6. Number of Smokers and Recent OC Users Among the 234 Cases and 1,742 Controls without Regard for Age (from Table V, p. 745)

Smoking Status		OC Use Yes	 No
	(MI	4	34
None	(Control	52	754
	(MI	3	79
1-24	(Control	51	566
	(MI	22	92
≥ 25	(Control	32	287

The rate-ratio estimates unadjusted for age are computed as follows:

	OC Use	
Cigarette Smoking	No	Yes
None	1.0 (Reference Category)	4/52 ÷ 34/754 = 1.71
1-24	79/566 ÷ 34/754 = 3.10	3/51 ÷ 34/754 = 1.30
≥ 25	92/287 ÷ 34/754 = 7.11	22/32 ÷ 34/754 = 15.25

Without the age adjustment, the rate-ratio estimates for non-OC users are similar to those given in Table VI, p. 746 (see Table 7, below). However, the estimates for OC users are much lower than those given by the authors. The rate-ratio for moderate smokers who use OC when unadjusted for age is smaller than the corresponding estimate for nonsmokers, which is also true in Table VI, p. 746. However, the rate-ratio estimate for heavy smokers who use OC is 15.25 when unadjusted for age and Table VI, p. 746, gives the figure as 39 (see Table 7 below); the authors' confidence limits do not include my figure of 15.

Table 7. Separate and Combined Effects of OC Use and Cigarette Smoking in Relation to MI: Age-Adjusted Rate-Ratio Estimates (95% Confidence Limits) (from Table VI, p. 746)

Cigarette Smoking	OC Use No	 Yes
None	1.0 (Reference Category)	4.5 (1.4-14.1)
1-24	3.4 (2.2 - 5.1)	3.7 (1.0-13.2)
≥ 25	7.0 (5.2 - 11.5)	39.0 (22-70)

The authors state that Table VI results are "derived from the date displayed in Table V," but this derivation is by no means clear. Further, the confidence limits are extremely wide for all OC use estimates (and even include 1.0 for moderate smokers) and for non-OC users who are heavy smokers. In addition, the numbers of OC users among the case (MI) women are extremely small, as the following table shows (derived from Table V, p. 745):

Cigarette Smoking	25-29	30-34	OC Users 35-39	40-44	45-49
None	0	0	0	1	3
1-24/day	1	1	1	0	0
≥ 25/day	3	8	3	5	3

These factors imply that the results presented in Table VI are probably not statistically reliable or valid.

Appendix D

Analysis of Data from Jick et al. (1978a)

Table 8. Number of Cases and Controls with No Predisposing Conditions According to OC Use (from p. 2,549)

	Control	MI	Totals
Non-OC User	49	7	56
OC User	14	23	37
Totals	63	30	93

$X^2_1 = 25.14, \quad P < .001$

Statistical Conclusion: There is a significant association between incidence of MI and OC use in women with no predisposing conditions.

Table 9. Number of Cases and Controls With Predisposing Conditions According to OC Use (from p. 2,549)

	Control	MI	Totals
Non OC User	14	19	33
OC User	0	3	3
Totals	14	22	36

$X^2_1 = 2.08 \quad .10 < P < .20$

Statistical Conclusion: There is a possible association between incidence of MI and OC use in women with predisposing conditions.

Table 10. Number of Cases and Controls with No Predisposing Conditions According to Cigarette Use (from Table 2, p. 2,549)

	Control	MI	Totals
Nonsmoking Now	72	4	76
Smoking	54	45	99
Totals	126	49	175

$\chi^2_1 = 34.45$, $P < .001$

Statistical Conclusion: There is a significant association between incidence of MI and smoking in women with no predisposing conditions.

Table 11. Number of Cases and Controls with Predisposing Conditions According to Cigarette Use (from Table 2, p. 2,549)

	Control	MI	Totals
Nonsmoking Now	14	5	19
Smoking	13	29	42
Totals	27	34	61

$\chi^2_1 = 9.68$, $.001 < P < .01$

Statistical Conclusion: There is a significant association between incidence of MI and smoking in women with no predisposing conditions.

Appendix E

Analysis of Data from Layde et al. (1981)

Table 12. Number of Smokers and OC Users Among the Deaths from SAH (from Table IV, p. 543)

	Deaths from SAH		
	Nonsmokers	Smokers	Totals
Controls (Non-OC Users)	1	5	6
Ever OC Users	2	12	14
Totals	3	17	20

$\chi^2_1 = .0187,\ .90 < P < .95$

Statistical Conclusion: In the population of women aged 35–44 who died from SAH, there is no association between OC use and smoking.

Table 13. Number of Smokers and OC Users Among the Deaths from Circulatory Disease (from Table V, p. 543)

	Deaths from Circulatory Disease		
	Nonsmokers	Smokers	Totals
Controls (Non-OC Users)	4	6	55
Ever OC Users	13	42	10
Totals	17	48	65

$\chi^2_1 = 1.17,\ .20 < P < .30$

Statistical Conclusion: In the population of women aged 35–44 who died from circulatory disease, there is no association between OC use and smoking.

Table 14. Number of Cases for Each Subgroup of Age and Smoking Status (from Table V, p. 543)

Age	Ever-Users	Non-OC Users
15-25(4)		
Nonsmokers	0	0
Smokers	1	0
25-34		
Nonsmokers	2	1
Smokers	6	1
35-44		
Nonsmokers	7	2
Smokers	18	3
45-		
Nonsmokers	4	1
Smokers	17	2

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SUMMARY OF INFORMATION CONCERNING

JEAN DICKINSON GIBBONS

Present Position:

Board of Visitors Research Professor of Statistics (1974-) and
Chairman of Faculty Committee for the Program in Applied Statistics (1975-) and
Professor of Mathematics (1976-), Graduate School, University of Alabama

Personal Data:

Age 43; Female; Excellent health; Married to John S. Fielden
Address: Box J, University of Alabama, University, Alabama 35486
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Positions Held:

1971-1974: Chairman, Department of Statistics and Quantitative Methods, College of Commerce and Graduate School of Business Administration, University of Alabama
1970-Present: Professor of Statistics, University of Alabama
1973 Summer: Research Associate, Stanford University
1968-1970: Associate Professor, Department of Statistics and Operations Research, University of Pennsylvania
1963-1968: Assistant Professor, University of Pennsylvania
1961-1963: Assistant Professor of Mathematics, University of Cincinnati
1962-1963: Consultant in Biostatistics, College of Medicine, University of Cincinnati
1958-1960: Instructor in Mathematics, Mercer University

Professional Recognition:

Fellow of American Statistical Association, Elected 1972
International Statistical Institute, Elected 1980
Outstanding Scholar of the University of Alabama, Elected 1980

Education:

A.B. magna cum laude, 1958, Duke University, Mathematics
M.A., 1959, Duke University, Mathematics
Ph.D., 1963, Virginia Polytechnic Institute, Statistics
Attended Columbia University full time, 1960-1961

Academic Honors:

Phi Beta Kappa (Junior year), A.B. magna cum laude
Phi Kappa Phi (graduate scholastic honorary)
Pi Mu Epsilon (mathematics honorary)
Chi Alpha Phi Chapter of Mu Sigma Rho (statistics honorary)
Beta Gamma Sigma (business honorary)

Biographical sketches in:

American Men and Women of Science
Dictionary of International Biography
Outstanding Educators of America
Personalities of the South
The World Who's Who of Women
Who's Who of American Women
Who's Who in the South and Southwest

Professional Society Memberships:

- American Statistical Association (A.S.A.)
- International Statistical Institute (I.S.I.)
- Alabama Chapter of A.S.A.

Current National Professional Activities:

- Committee on National Statistics of the National Research Council of the National Academy of Sciences, 1980-83
- Executive Committee of the A.S.A. Section on Statistical Education, 1982-84 (nationally elected office)
- A.S.A. Advisory Committee on Continuing Education, 1981-83
- Southern Regional Education Board Committee on Statistics, Representative of The University of Alabama, 1972-; Chairman, 1980-82; Treasurer, 1978-80
- Director at Large of Mu Sigma Rho, 1979-82
- Committee on Affirmative Action of the Conference Board of the Mathematical Sciences, 1977-
- Reviewer of grant proposals submitted to the National Science Foundation, 1976-
- Editorial Collaborator for various statistics journals, including (at various times) Journal of the American Statistical Association, Communications in Statistics, The American Statistician, Technometrics, Biometrics, The Annals of Statistics, Sankhya, Journal of Quality Technology, Journal of Educational Research, Journal of Educational Statistics

International Professional Activities:

- Member of the International Statistical Institute, Elected 1980
- Co-Editor of the Proceedings of the International Insurance Seminar, 1978-80; Participant in meetings, 1978-80
- Senior Fulbright-Hays Scholar, Indian Statistical Institute, 1973
- Delegate to NATO Statistical Conference, Greece, 1972
- Delegate to VI International Biometrics Congress, Australia, 1967

Selected Past Professional Activities:

- Associate Editor, The American Statistician, 1972-80
- Executive Committee of the A.S.A. Section on Statistical Education, 1977-80 (nationally elected office)
- A.S.A. Advisory Committee to the U.S. Office of Management and Budget, 1976-80
- Chairman of the Editorial Search Committee of A.S.A., 1979
- Board of Directors of A.S.A., 1975-77 (nationally elected office)
- Chairman, A.S.A. Committee on Meetings, 1975-77
- Program Chairman for the 1976 annual meeting of the A.S.A.
- Advisory Board of the National Cancer Institute, DHEW, 1974-76
- President, Alabama Chapter of the A.S.A., 1974-76; Vice-President, 1972-74
- Chairman, National Committee on Women in Statistics of A.S.A., 1972-76
- Lecturer for Visiting Lecturer Program of A.S.A. and I.M.S., 1973-75
- Representative-at-Large to the Council of A.S.A., 1972-74 (nationally elected office)
- Constitution Committee of A.S.A., 1972-74
- Committee on Elections of A.S.A., 1974
- Recent Publications Editor of The American Statistician, 1970-73
- Co-Director, CBMS and NSF Conference on Multivariate Statistical Analysis, Summer 1973
- President, Phi Beta Kappa Chapter, University of Pennsylvania, 1967-69

PUBLICATIONS OF JEAN D. GIBBONS

Books:

Nonparametric Statistical Inference, McGraw-Hill Book Co., New York, 1971, pp. 306. This graduate level reference and textbook is included in the McGraw-Hill Series in Probability and Statistics and also in the International Student Edition Program.

Nonparametric Methods for Quantitative Analysis, Holt, Rinehart and Winston, Inc., New York, 1976, pp. 463. This graduate level textbook, reference or handbook is included in the International Series in Decision Processes, edited by Ingram Olkin.

Selecting and Ordering Populations: A New Statistical Methodology, co-authored with Professor Ingram Olkin, Stanford University, and Professor Milton Sobel, University of California, Santa Barbara, John Wiley & Sons, New York, 1977, pp. 569. This is the very first book on this topic; it is a volume in the Wiley Series in Probability and Mathematical Statistics.

Concepts of Nonparametric Theory, co-authored with Professor John W. Pratt, Harvard University, Springer-Verlag, New York, 1981, pp. 462. This graduate and research level book is included in the Springer Series in Statistics.

Refereed Publications:

"On the Equiprobability of Two Rank Orders," Abstract with H. A. David, Annals of Mathematical Statistics, 1963, Vol. 34, 357.

"Effect of Non-Normality on the Power of the Sign Test," Journal of the American Statistical Association, 1964, Vol. 59, 142-148.

"On the Power of Two-Sample Rank Tests on the Equality of Two Distribution Functions," Journal of the Royal Statistical Society, B, 1964, Vol. 26, 293-304.

"A Proposed Two-Sample Rank Test: The Psi Test and its Properties," Journal of the Royal Statistical Society, B, 1964, Vol. 26, 305-312.

"A Correlation Measure for Nominal Data," The American Statistician, December 1967, Vol. 21, 16, with Kenneth H. Ives as first author.

"Correlation Coefficients between Nonparametric Tests for Location and Scale," Annals of the Institute of Statistical Mathematics, 1967, Vol. 19, 519-526.

"Mutually Exclusive Events, Independence and Zero Correlation," The American Statistician, December 1968, Vol. 22, 31-32.

"Estimation of the Number of Critical Sites in Limited Genome Expression During Viral Infection of Bacteria," *Biometrics*, 1969, Vol. 25, 537-544, with Samuel Litwin as second author.

"Properties of the Percentile Modified Rank Tests," *Annals of the Institute of Statistical Mathematics Supplement*, 1970, Vol. 6, 95-114, with Joseph L. Gastwirth as second author.

"A Unified Approach to Hypothesis Testing," *Estadistica*, 1970, Vol. 28.

"A Distribution-Free Two-Sample Goodness-of-Fit Test for General Alternatives," *British Journal of Mathematical and Statistical Psychology*, 1972, Vol. 25, 95-106.

"On the Design of a Random Alarm," *Journal of Physics - E: Scientific Instruments*, 1972, Vol. 5, 634-637, with Samuel Litwin as second author.

"Comparisons of Asympototic and Exact Power for Percentile Modified Rank Tests," *Sankhya*, *B*, 1973, Vol. 35, 15-24.

"A Question of Ethics," *The American Statistician*, April, 1973, Vol. 27, 72-76.

"Estimation of the Unknown Upper Limit of a Uniform Distribution," *Sankhya*, B, 1974, Vol. 36, 29-40.

"Reporting P-values as an Aid to Decision Making," competitive paper in *Proceedings of Southwest Conference Meeting of American Institute for Decision Sciences*, 1974, Cincinnati, Ohio, 18-20.

"The Visiting Lecturer Program," Letter in *The American Statistician*, 1974, Vol. 28, 35.

"Simultaneous Estimation of the Unknown Upper and Lower Limits in a Two-Parameter Uniform Distribution," *Sankhya*, B, 1974, Vol. 36, 41-54, with Samuel Litwin as second author.

"P-values: Interpretation and Methodology," *The American Statistician*, 1975, Vol. 29, 20-25, with John W. Pratt as second author.

"The Status of Statistics in the Seventies," Letter in *The American Statistician*, 1976, Vol. 30, 150.

"Comparing the Mean and the Median as Measures of Centrality," *International Statistical Review*, 1977, Vol. 45, 63-70, with Gordon R. Stavig as first author.

"Baseball Competitions—Do They Play Enough Games?," *The American Statistician*, 1978, Vol. 32, 89-95, with Ingram Olkin and Milton Sobel.

"Parameter Measures of Skewness," *Communications in Statistics*, *B*, 1979, Vol. 8, 161-167, with William G. Nichols as first author.

"An Introduction to Ranking and Selection," *The American Statistician*, 1979, Vol. 33, 186-195, with Ingram Olkin and Milton Sobel.

"Quantitative Coefficients for Selecting a Measure of Central Location," with Gordon R. Stavig. Chapter 18 in *Sociological Methodology 1980* (K. F. Schuessler, Ed.), Jossey-Bass, San Francisco, pp. 545-558, 1980.

"A Subset Selection Technique for Scoring Items on a Multiple Choice Test," *Psychometrika*, 1979, Vol. 44, 259-270, with Ingram Olkin and Milton Sobel.

"Organizations for Statistical Consulting at Colleges and Universities," *The American Statistician*, 1980, Vol. 34, 140-145, with R. J. Freund.

"Selection Procedures: A New Statistical Methodology and its Applications for Marketing Research," *Journal of Marketing Research*, 1981, Vol. 18, 449-455, with Oded Gur-Arie.

"Brown-Mood Median Test." An entry in the *Encyclopedia of Statistical Sciences*, Volume 1 (Norman L. Johnson and Samuel Kotz, Eds.), John Wiley & Sons, New York, 1982.

"Methods for Selecting the Best Process," *Journal of Quality Technology*, 1982, Vol. 14, No. 2.

Other Publications:

Author of statistics section, pp. 106-108, of *Nontraditional Careers for Women* by Sarah Splaver, Julian Messner, New York, 1973.

Editor, *Roster of Women in Statistics*, The American Statistical Association, Washington, D.C., July 1974.

"Nonparametric Statistical Methods," by Myles Hollander and D. A. Wolfe, John Wiley, New York, 1973, Review in *Technometrics*, 1974, Vol. 16, 477-478.

Quality of Scientific Evidence in FDA Regulatory Decisions, Hearing before a Subcommittee of the Committee on Government Operations, House of Representatives, Ninety-Fifth Congress, U.S. Government Printing Office, Washington, D.C., 1978.

Technical Reports published by Johns Hopkins University, University of Georgia, and University of Michigan.

Selected Colloquia Presentations:

Virginia Polytechnic Institute, February 1976
SREB Summer Research Conference, June 1977
North Carolina State University, October 1977
University of South Carolina, February 1978
Winter Simulation Conference, December 1978, Miami
Naval Postgraduate School, June 1979, Monterey
A.S.A. Short Course, August 1979, Washington, D.C., with Robert E. Bechhofe
Shanti S. Gupta, and Ingram Olkin, available on videotape from the A.S.A.
A.S.A.-A.S.Q.C. Fall Technical Conference, October 1979, Minneapolis

Statement of Katherine McDermott Herrold, M.D.

My name is Katherine McDermott Herrold. I am a certified pathologist and am presently retired from my most recent position as medical director of the United States Public Health Service.

I received my medical degree from Women's Medical College of Pennsylvania in Philadelphia in 1948. From 1948 through 1949 I interned at George Washington University Hospital in Washington, D.C. Between 1952 and 1955 I was the chief medical officer at the Federal Reformatory for Women in Alderson, Va. Between 1955 and 1957 I was a resident in clinical pathology, and between 1957 and 1959 I was a resident in pathological anatomy, both of these positions being at the National Institutes of Health in Bethesda. Between 1959 and 1971 I was a laboratory pathologist at the National Cancer Institute in Bethesda, Maryland.

I am a member of numerous professional societies including the American Society of Clinical Pathologists, the College of American Pathologists, the International Academy of Pathology and the American Association for Cancer Research. I am a member of the honorary medical society of Alpha Omega Alpha.

I have published papers in the scientific literature dealing with pathology and cancer research.

Those who assert that cigarette smoking causes lung cancer rely on the data contained in several prospective studies, including one whose data was first presented by Dr. Harold F. Dorn in 1958. That study reported a statistical association between smoking and lung cancer. However, Dr. Dorn recognized the grave danger of relying upon death certificates for lung cancer diagnoses without pathological confirmation. It was my privilege to work with Dr. Dorn on this project in providing the clinical and pathological review of the data.

The Dorn study involved a total of almost 300,000 U.S. veterans who were policyholders of U.S. Government life insurance. I reviewed the clinical and pathological materials available from the 2,241 patients with the diagnosis of lung cancer on their death certificates. Histologic material was available for review in about 65% of those cases. Extremely important from a biological standpoint is the fact that only a small percentage of even heavy cigarette smokers develop lung cancer.

The frequency of the histologic types of cancer was also established. Of the 472 patients who were "current cigarette smokers" at the time of their deaths, and for whom histologic sections were available for my review, I found no correlation between the various histologic types of lung cancer and the amount of tobacco smoked. Further, the age at death from lung cancer was not related to the age at which smoking started, the number of years smoking, or the number of cigarettes smoked per day.

These findings were in agreement with those of R.D. Passey some ten years earlier. Dr. Passey wrote that it could be said without hesitation that the age at death from lung cancer is not that which is associated with chemical carginogenesis. Passey's findings, as well as those of Doll and Hill, were almost identical to mine - that the age of diagnosis of lung cancer is not related to the age at which the patient started to smoke, the length of time smoking, or the amount smoked.

Those who make claims against smoking assert that there is a dose response relationship between smoking and lung cancer; that is, the risk of developing lung cancer increases with the number of cigarettes smoked and the earlier one begins to smoke the greater the risk. My findings, like those of Dr. Passey before me, suggest otherwise.

In recent years, there have been reports of a so-called "epidemic" of lung cancer, which some have attributed to increased tobacco consumption. However, scientists have noted that this apparent increase may well reflect changes in the detection rate rather than in the actual incidence of the disease. The diagnostic problem is exacerbated by reason of "detection bias" among smokers, i.e., the fact that smokers undergo a more rigorous examination for lung cancer than non-smokers. Studies reporting that lung cancer occurs more frequently in smokers than in non-smokers all *assume* that the non-smokers are studied with an equal frequency and intensity as the smokers. However, Professor Alvan Feinstein of the Yale Medical School found this not to be the case at various institutions, including the Yale-New Haven Hospital. Hence, he concluded in 1974 that "cigarette smoking may contribute more to the diagnosis of lung cancer than it does to producing the disease itself."

The various anomalies found by myself and others in the population studies combined with the problems associated with diagnosing lung cancer, lead me to the conclusion that more research is needed in this field before we can accept as proven the theory that smoking causes lung cancer.

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24 February 1982

To the Committee:

This is my statement relative to H.R. 4957

Arthur Furst

Arthur Furst, Ph.D.
Distinguished University Professor
(Emeritus)

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STATEMENT OF ARTHUR FURST, Ph.D.

As a citizen and a resident of California since 1919, I applaud Congressman Waxman's continuing efforts to improve the health of the American people. His concern is certainly appreciated. I question, however, the scientific basis of his proposal (H.R. 4957) to amend the Public Health Service Act and the Federal Cigarette Labeling and Advertising Act.

As the attached copy of my curriculum vitae shows, I hold the only Distinguished University Professorship (Emeritus) at the University of San Francisco. I am the Director (Emeritus) of the Institute of Chemical Biology (USF), and I have been a professor of pharmacology at Stanford University School of Medicine and a (visiting) clinical professor of pathology at the College of Physicians and Surgeons of Columbia University. I am listed in Who's Who in the World and World Who's Who in Science. I am a consultant (temporary) to the World Health Organization. In fact, I was a member of the international working group that wrote IARC Monographs Volume 2 and 23 on Evaluation of the Carcinogenic Risk of Chemicals to Humans. I am currently organizing an international conference on the toxicity and reproductive hazards of heavy metals. I also consult frequently with governmental agencies and private industries on a variety of environmental health problems.

I have read H.R. 4957 with great interest, for I have spent over thirty years in scientific research, much of it studying the questions of smoking and health. My original laboratory work on carcinogenesis antedates the first Surgeon General's Report on Smoking and Health. Over the years, I have studied the possible effects on animals of both whole smoke and various smoke components. I have also investigated the carcinogenicity of a wide variety of substances, particularly the heavy metals, and I have carefully

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monitored the world literature on experimental carcinogenesis. Perhaps this background will help explain my concerns about H.R. 4957, which makes such flat, dogmatic statements about a scientific area which I know to be fraught with uncertainties.

A Congressional finding that "cigarette smoking is the number one cause of lung cancer" implies a scientific certainty that I, as a scientist, believe to be unwarranted. My skepticism arises primarily from my extensive knowledge of and first-hand experience with animal experiments on tobacco smoke and lung cancer.

For many years, I tried to induce lung cancer in animals with cigarette smoke, with no success, despite the most sophisticated smoking machines available. Not only were my colleagues and I unsuccessful, but every other investigator who attempted to induce lung cancer in animals by inhalation of fresh smoke also failed.

There have been a very small number of published reports of lung cancers occurring in experimental animals during smoke inhalation experiments. Anyone attempting to interpret these as showing that smoking causes lung cancer must understand that animals, like humans, do spontaneously develop lung cancer even in the absence of any suspected carcinogen. The key question that an investigator must ask himself is whether any cancers that develop in his animals are actually a result of the experimental exposure. For example, rodents have a fairly high spontaneous rate of adenocarcinoma (malignant tumors) and adenoma (nonmalignant tumors) development. I have examined the data on the reports of increased adenocarcinoma in smoke-exposed animals and have found no difference between the incidence reported in the experimental animals and the normal baseline, spontaneous rate.

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The lung cancer cell type traditionally associated with human smoking is squamous cell carcinoma. The spontaneous rate of this cancer is considerably lower than that of adenocarcinoma in rodents, but it too does occur naturally in these animals. There have been very few reports of a squamous cell cancer in smoke-exposed animals, and generally the authors made no comment on these findings. I assume that they concur with me that they were seeing nothing more than spontaneous production.

One of the animal experiments that has received the most attention is that on beagle dogs conducted by Auerbach et al. Twelve years ago, they claimed to have succeeded in producing lung cancer in their beagles, but because their experimental methods were seriously flawed, I consider their conclusions to be unfounded. Because the Auerbach experiment occasionally resurfaces as virtually the only example of a positive inhalational study result, I feel compelled to repeat some of the criticisms of that study that I voiced in 1972. Any time this experiment is praised, there seems to be no emphasis on the fact that the dogs received the smoke through a tracheostoma. This in no way resembles human smoking. Unless the pictures I have seen are wrong, the smoke was initially forced into the lungs through the trachea by use of a pump! This was without regard to the normal respiration rate of the dogs. Further, several of the dogs died from infections, aspiration of foreign material, etc., which is unusual in a well controlled experiment. This experiment has no meaningful relationship to the human experience, and any reports of tumors should be considered in light of the fact that the experimental data were not made available to an independent panel for evaluation, as requested. Also, since beagle dogs do get lung cancer spontaneously, a discussion of this fact would have been useful to readers interested in evaluating the Auerbach work.

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The design and conduct of meaningful animal experimentation require a great deal of sophistication. I have reviewed a vast number of research proposals and reports in the past 30 years, and I can assure you that a large number of the experimental designs or the conclusions drawn from the experimental data do not stand up to rigorous scientific scrutiny. One of the simplest requirements that is often overlooked is the need to select from litter mates the controls and the experimental animals. Even with exactly the same strain of mice, the animals' spontaneous rate of tumor appearance will vary from one supplier to another; this can cause serious problems with the interpretation of the data. Thus, even if positive results are obtained in one experiment, a replication using animals from a different litter may yield nothing unusual.

It is also extremely important to use "clean" animals. Parasites or infectious diseases in the animals can seriously affect experimental results, even if the animals are successfully treated before the experiment begins. For example, we can't be sure what effect the parasite infestation in Dontenwill's Syrian Golden hamsters had on his inhalation study results. Similarly, the possibility that Auerbach's beagles had lung worms causes concern. In my own work, I always try to avoid using unclean animals by checking them very carefully before beginning an experiment. If I discover later that my animals are not clean, I terminate the experiment and start again with new animals. That is the only way that I can feel certain that the infestation or infection will not affect the experimental findings.

In summary, based on my own research and familiarity with the literature, I have concluded that no reliable, reproducible animal studies have shown that the inhalation of cigarette smoke causes lung cancer. I might add that skin-painting experiments are inappropriate for studying the question

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of tobacco smoke and cancer. We must insist that animal experiments simulate, as closely as possible, the human experience — and skin-painting, as well as certain other experimental techniques, fail to mimic adequately human inhalation.

Various groups convinced of smoking's causal role in lung cancer development have attempted to ignore or explain away the negative results in animal inhalation studies. For example, I was once challenged in a scientific meeting about the mice I used in my smoke inhalation studies. The individual argued that this strain could not develop lung cancer, and that is why I failed. But this is not true, because we have documented well the production of lung cancer in these animals. Over a period of years we published a series of papers describing our technique of instilling carcinogens in the lungs of mice, the sensitivity of various strains of mice to carcinogens, and the response of the animals to different hydrocarbons. Yes, development of all histological types of lung cancer is common in animals treated with various carcinogens. It is even possible to rate experimentally the potency of carcinogens as inducers of lung cancer.

In experimental carcinogenesis, replication by independent investigators is essential to establish the validity of the findings. Other researchers have been successful in replicating my experimental production of lung cancers. For example, in the latest issue of Cancer Research (vol. 41), Henry, et al., published a complete confirmation of my research. They used the same technique, strain of mouse, chemical, and reported the same time of lung cancer appearance. It is in this context of successful lung cancer induction techniques that the failures of smoke inhalation studies should be judged.

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In general, the major health claims against cigarette smoking are based mainly on epidemiological studies. From the standpoint of science, epidemiological studies can only point out statistical associations between a factor (such as smoking) and disease; they cannot prove a causal relationship. Yet, the findings in H.R. 4957 are stated as causal relationships, as though all is proven. Thus, as a scientist with over 30 years experience in laboratory research, I must disagree with those findings on the grounds that they are overstatements of the scientific knowledge — all is not proven in the smoking and health area.

I heartily agree with the recommendation that more research must be funded and conducted. Yet, I must emphasize that negative results can also be very important. Scientific knowledge (in this case, of lung cancer causation) advances only by publication of both successes and failures. This is why I have been troubled that so little information is given in the reports by the Surgeon General about the negative experimental results in the lung cancer area. A case in point is Dr. A. Wehner's work which showed that hamsters, as a result of their exposure to cigarette smoke, not only failed to develop lung cancer, but also lived longer! Yes, the requirement that the Office on Smoking and Health "collect, analyze, and disseminate" smoking and health information is excellent if both positive and negative findings will be reported. Only with this unbiased approach can the needs of both science and the public in general be met. I feel compelled to say that past performance by official agencies has been characterized by highly selective reporting.

I am particularly disturbed by the latest (1982) Report of the Surgeon General Health Consequences of Smoking. Like so many of its predecessors, it often omits references to material contradictory to the positions it has adopted. Why the omissions? Why should a government document selectively present only that part of the evidence that supports its position?

In general, this Report provides no new information that would make me change my mind about the lack of good scientific information on smoking and health.

I agree with the Report's recommendation that major research efforts should be devoted to elucidating the mechanisms of lung cancer development. At this point, we have a number of theories, not proof, and in many instances not very good theories.

The writers of the Report assume that the case against cigarette smoking is proven, and that the lack of knowledge about the mechanisms involved is only the finishing touch — not really essential to prove the point. I cannot disagree more strongly. Unlike the writers of the Surgeon General's Report, I believe that until we have that proof, any conclusions regarding causation are premature.

I disagree with the Report's discussion of the animal inhalation experiments using Syrian golden hamsters (many of them conducted by Dontenwill, et al.) A summary section of the Report says that "studies on smoke inhalation with the hamster now appear suitable" for evaluating the "tumorigenic potential" of cigarette smoke. This seems highly incompatible with the Report's earlier comment that "why these inhalation experiments with hamsters did not induce carcinoma of the lung remains to be elucidated." Lung cancer can be induced in hamsters, by using a variety of substances. Also, significant amounts of cigarette smoke do reach the hamsters' lungs. So why don't the hamsters get lung cancer after exposure to tobacco smoke?

The 1982 Report concludes that inhalation studies have basically failed to induce lung cancer in animals, but then it recommends that further research efforts in this area should receive limited priority. I find that incomprehensible. The animal data are significant negative evidence. They basically contradict the popular interpretation of the epidemiological data. Why? We must have good research to find out.

I am not in any way advocating smoking; as a scientist, I am concerned with presenting the best information to the public, both positive and negative. I am also very concerned that if all the diseases noted in this bill are assigned by federal law to smoking, there will be a severe decline in research on and attempted control of many known or suspected environmental causes of disease. For example, this federal law can only have an adverse effect on efforts to clean up our environment, to continue smog controls in cars, and to remove the particulates and noxious gases in the atmosphere.

I have lectured throughout California in programs on the Biology of Cancer sponsored by the American Cancer Society. Such contact has led me to believe that the public is well informed on what the preamble in H.R. 4957 refers to as the "dangers of smoking." Every package of cigarettes, every magazine ad, and every billboard ad carries the Surgeon General's warning. Furthermore, the American Lung Association, the American Cancer Society, and the March of Dimes are continually funding radio and TV announcements against smoking.

I cannot agree, therefore, with this legislative requirement that the government spend even more time and effort to inform the public as though they are ignorant of all the warnings that have been broadcast over the past nearly 20 years. It seems to me that the public is indeed well informed, and

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that perhaps they have simply decided that they wish to make their own decision. I would prefer to have these millions of dollars spent on good research, and not on more publicity campaigns.

Again, I respectfully suggest that Congress should encourage good research; I have been advocating this for 30 years. If we had received adequate funding of good research years ago, then many of the questions raised today might have been answered. Science, not publicity campaigns, will solve our problems.

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CURRICULUM VITAE OF ARTHUR FURST

PERSONAL:

Born Minneapolis, Minnesota, December 25, 1914. Moved to California, 1919. Married, four children.

EDUCATION:

Los Angeles City College	1932-35	Psych., Chemistry	A.A.
University of California at Los Angeles (UCLA)	1935-37	Chemistry, Physics Education	A.B.
UCLA	1940	Chemistry, Math.	M.A.
Stanford University	1948	Chemistry	Ph.D.

PROFESSIONAL EXPERIENCE: I

1937-39	Teaching Assistant University of California at Los Angeles
1939-40	Teacher, Science and Mathematics Pacific Military Academy, Culver City, California
1940-47	Instructor, Chemistry Department San Francisco City College
---	During World War II, part time at: University of California War Training; San Francisco State College; University of San Francisco
1947-49	Assistant Professor of Chemistry University of San Francisco
1949-52	Associate Professor of Chemistry University of San Francisco
1949-82	Research Associate Mount Zion Hospital, San Francisco
1952-61	Lecturer, Chemistry Department University of San Francisco
1952-57	Associate Professor of Pharmacology (Medicinal Chemistry) Stanford University School of Medicine

1957-61 Professor of Medicinal Chemistry, Pharmacology Dept.
Stanford University School of Medicine

1961-81 Director, Institute of Chemical Biology
Professor of Chemistry
(Emeritus 1981) University of San Francisco

1976-79 Dean, Graduate Division
University of San Francisco

1979-81 Distinguished University Professor
(Emeritus 1981) University of San Francisco

CONCURRENT TITLES: 1978-1981 (retired)

a) Consulting Professor of Pharmacology
Stanford University School of Medicine

b) Research Associate
University of Calif. Medical Center, San Francisco

c) Guest Scientist
Lawrence Berkeley Laboratories, Univ. Calif. Berkeley

PROFESSIONAL EXPERIENCE: II

During sabbatical leaves of absence:

1946 Visiting Scientist, Radium Institute, Paris

1969 Clinical Professor, Pathology
College of Physicians and Surgeons,
Columbia University, New York

1974 Visiting Scholar, University of Washington, Seattle, and Visiting Fellow, Battelle Seattle Research Center, Washington

PROFESSIONAL EXPERIENCE: III

Guest lecturer at many universities throughout the world

Speaker at many symposia, worldwide, and at Gordon Conferences

Consultant to various industries and government agencies

MEMBER OF:

American Association for the Advancement of Science
  (Fellow 1956)
American Association for Cancer Research
American Chemical Society
  (Member, Executive Board, three years)
American College of Toxicology (Secretary and Council
  Member)
American Public Health Association
American Society of Pharmacology and Experimental Therapeutics
California Association of Chemistry Teachers
  Organizer, First President
New York Academy of Science (Fellow 1956)
Phi Lambda Upsilon (Honorary Scholastic Society)
Royal Society of Medicine (Associate Member)
Society of Environmental Geology and Health
Society Sigma Xi (Life Member)
Society of Toxicology
Western Pharmacology Society (Charter Member)

LISTED IN:

Who's Who in the World
Who's Who in America
Who's Who in the United States
American Men of Science (Physical and Medical)
International Biography
Who's Who in American Education
Who's Who in College and University Administration
Who's Who in Health Care
Who's Who in the West
World Who's Who in Science
International Bibliography--Men of Achievement (Cambr.,Eng.)
National Register of Prominent Americans

PUBLICATIONS:

Over 200 publications in the fields of:

1) Cancer Research
   a) Carcinogenesis
   b) Chemotherapy (including viruses)

2) Organic Synthesis

3) Psychopharmacology

MISCELLANEOUS:

1970 Consultant (temporary) World Health Organization

1973 Seventh Distinguished Teaching Award
University of San Francisco

1975 American Public Health Association Task Force
on Environmental Problems

1976 National Research Council. Commission on Natural
Resources. Board of Mineral Resources

1978 Committee to Review Standards, Mine Safety and Health
for Secretary of Labor

MEMBER OF EDITORIAL BOARD:

Current Contents/Life Sciences
Journal Combustion Toxicology
Journal Environmental Pathology and Toxicology (Book review editor)
Toxicology and Applied Pharmacology

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Statement of Richard J. Hickey, Ph.D.

I am Richard J. Hickey, a senior research investigator in the Department of Statistics of The Wharton School, University of Pennsylvania, Philadelphia. I received a Ph.D. degree in biophysical chemistry and microbiology at Iowa State University, Ames, Iowa, in 1941. Thereafter, I performed research and development work on antibiotics, vitamins and other biochemicals. In the mid-1960's, I became involved in research on the air pollution/public health problem at the University of Pennsylvania. Since 1973, I have been investigating the relationships of environmental chemicals and other variables to chronic disease risk at the University's Wharton School. Many of the journal articles and book chapters published, of which I was author or co-author, pertain to environmental pollution.

The following comments concern both H.R. 4957 and S. 1929. A basic criticism of both proposed acts is the implicit assumption that "findings" (1), (2), (3), and (4), which are used to justify both the labeling statements and the proposed actions, are known and proven. However, there are serious scientific questions about the scientific bases for the claims that cigarette smoking causes the various conditions associated statistically with smoking, such as lung cancer, heart disease, and low birth weight.

Documentary scientific evidence establishing causality is not given in the proposed bills. However, it is clear from the wording of the "findings" that they rely heavily upon the alleged causality evidence presented in some publications and reviewed in recent U.S. Surgeon Generals' reports on smoking and health. Unfortunately, these reports do not represent an even-handed review of the available evidence. There are several reasons for rejecting the conclusions of these reports, including:

(A) Selective Reporting or Selective Exclusion. Selective reporting is present in the Surgeon Generals' reports. Sources that conform to the pre--determined smoking-causality position of the reports are cited extensively, while studies that challenge or discredit this position are often selectively excluded. Warnings of the misuse of statistics have also been excluded. The Surgeon Generals' reports have the characteristics of briefs for the "prosecution" only; the "defense" has been largely omitted even though these reports should have been unbiased.

(B) Uses and Misuses of Statistics. The Surgeon General's report of 1964 asserts (p. 182): "Statistical methods cannot establish proof of a causal relationship in an association. The causal significance of an association is a matter of judgment which goes beyond any statement of statistical probability." These two statements lie at the root of the problem. The first sentence is a correct statement; causality cannot be established by statistical associations. This is a standard caveat that is taught in elementary statistics courses. The second statement is false. Subjective judgment or personal opinion cannot be used to interpret the causal implications of a statistical association, no matter how strong and no matter what level of significance. Judgment and opinion do not constitute substantive evidence.

It should be recognized that statistical associations are mathematical quantities. When properly obtained, they may suggest possible causal relationships, but they can never prove such relationships. Human conditions such as cancer, heart disease, spontaneous abortions, stillbirths and birthweight deficiencies are all basically biological and biochemical problems, not mathematical or statistical problems. When ill, one visits his physician, not his statistician.

Properly used, statistical methods are capable of rejecting an hypothesis to the extent that it is incompatible with observed data, but statistics alone can never establish that an hypothesis is certainly true. This fact is widely misunderstood. However, recent Surgeon Generals' reports have rather routinely inferred causality from statistical association, e.g., that cigarette smoking causes lung cancer if the two variables are positively associated in the statistical sense.

Statistical associations may be due to chance or to dual influences of some third factor. For example, it was reported in 1952 that "the[illegible]s ... a significant correlation in the United States between the population to physician ratio and the population to bed ratio and the number of reported deaths from cancer of the lung." [R.H. Rigdon and H. Kirchoff, "A Consideration of Some of the Theories Relative to the Etiology and Incidence of Lung Cancer," Texas Rept. Biol. Med. 10, 76-91 (1952) (p. 88)]. Surely no one would conclude from this correlation that physicians "cause" lung cancer.

It is known that cigarette smokers tend to be coffee drinkers; there is a positive correlation between measures of cigarette and coffee consumption in some populations. Is it to be concluded, therefore, that smoking "causes" coffee drinking, or that coffee drinking "causes" smoking?

Another fallacy is the presumption by some that non-random samples are not biased. This belief is especially hazardous when working with genetically heterogeneous populations such as of humans. For example, some studies that employ self-selected samples such as those of heavy smokers, light smokers, non-smokers, and ex-smokers are likely to be biased. This point was addressed by T.D. Sterling

["The Statistician vis-a-vis Issues of Public Health," American Statistician 27, 212-216 (1973) (pp. 212-213)]: "The manner of selecting populations to study the effects of smoking has been criticized by some of the world's most respected statisticians but to little avail (Berkson, 1958, 1960; Fisher, 1957, 1958 Mainland, 1956; just to mention a few). Of concern to statisticians has been the major reliance on 'volunteers' in most of these studies and the consequent multiple sources of serious bias. Ordinarily, results from a study such as Doll's in which over 30 per cent of the queried population did not participate would be considered, if at all, with greatest suspicion, especially if, as is freely admitted, the responders were quite different from the reference population. (Doll, 1964). Similarly, the data collected by volunteers of the American Cancer Society and used to link smoking to a variety of diseases stem from a study population (also of volunteer families) that dramatically differs in most significant comparable measures from the U.S. population (Sterling, 1971a, 1972a)."

(C) The Genetic/Constitutional Alternative. In the late 1950's, Sir Ronald Fisher, a former President of the Royal Statistical Society, suggested the following alternative to the smoking-causality hypothesis: "Cigarette smoking and lung cancer, though not mutually causative, are both influenced by a common cause, in this case the individual genotype." The problem of resolving the conflict between the smoking-causality hypothesis and the genetic/constitutional hypothesis was recently reviewed in depth by P.R.J. Burch ["Smoking and Lung Cancer: The Problem of Inferring Cause (With Discussion," Journal of the Royal Statistical Society, Ser. A (General), Vol. 141, Part 4, pp. 437-477 (1978)]. Burch concluded (p. 476): "The discussion has allowed me to undermine one causal model and it has revealed divergent opinions that might surprise those nurtured on the reports of the Royal College of Physicians and the Surgeon

General of the United States. I am also happy to record that the constitutional theory, proposed by a former President of this Society, the late R.A. Fisher, is alive and well."

To this date the genetic/constitutional hypothesis has not been discredited. It has, however, been denigrated subjectively and widely ignored. Despite the fact that Burch's paper appeared in a major statistical journal, it has not been cited in any Surgeon General's report. Further, the constitutional hypothesis has been "criticized" by some who would seem to equate criticism with rejection. Some criticism has been of the "do not like" variety.

Unless the genetic/constitutional alternative hypothesis is rejected on valid scientific grounds, it would be risky to base public policy upon the smoking-causality hypothesis.

If more anti-smoking legislation is passed and if eventually it is established that much habitual smoking is in fact symptomatic of an underlying genetic trait that both influences smoking behavior and contributes to increased risks of developing those diseases associated statistically with smoking, then the U.S. government would be placed in a totally untenable position. The prospect that R.A. Fisher, considered by some to be the father of modern statistics, has been correct all along is very real and cannot be ignored.

(D) Air Pollution and Chronic Disease. In addition to the omission of publications such as that of Burch, the Surgeon Generals' reports have also extensively excluded from consideration published epidemiological reports on the complex relationships between a number of common urban air pollutant chemicals and mortality rates for several categories of cancer, heart disease, and other causes of death in U.S. cities.

Some reports showed that concentrations of several common air pollutants such as sulfur dioxide, sulfate, nitrogen dioxide, and trace metals in the air of 38 cities were significantly associated statistically with lung cancer mortality rates for these cities. In fact, over 70% of the variance in lung cancer mortality rate was explained statistically by air pollutant characteristics of these cities. Further, over 50% of the variances in mortality rates for several other cancer classifications were also explained statistically by air pollution data. Also, over 50% of the variances in mortality rates for certain heart disease classifications was explained statistically by air pollutant concentration data for these cities. Significant relationships were also found between air pollutant concentration data and mortality rates for infants under one year of age and mortality rates for congenital malformations. [See, for example, R.J. Hickey et al., "Ecological Statistical Studies Concerning Environmental Pollution and Chronic Disease," IEEE Transactions on Geoscience Electronics, Vol. GE-8, 186-202 (1970); R.J. Hickey, "Air Pollution," pp. 189-212, in: Environment: Resources, Pollution & Society (W.W. Murdoch, Ed.), Sinauer Associates, Stamford, Conn., 1971; R. Mendelsohn and G. Orcutt, "An Empirical Analysis of Air Pollution Dose-Response Curves," Journal of Environmental Economics and Management, Vol. 6, 85-106 (1979)].

(E) Comments on Four "Findings" of H.R. 4957 and S. 1929. The following comments refer to "findings" (1), (2), (3), and (4) that appear on page 2 of both bills. The greatest detail is offered regarding finding (4) which pertains to pregnancy, prematurity, low birth weight, and related problems of infant health.

Finding (1). H.R. 4957 states that "cigarette smoking is the largest preventable cause of illness and premature death in the United States and is associated with the unnecessary deaths of over three hundred thousand Americans annually." This statement pertains in large part to deaths from cancer and from cardiovascular diseases. However, it says nothing about the constitutional hypothesis nor about epidemiological and other evidence regarding the relation of air pollution to chronic disease.

Finding (2). S. 1929 states that "smoking is the primary cause of lung cancer" in the United States, and "is associated with other cancers." This claim has been made in various ways by the Surgeon Generals' reports. However, as stated previously, this claim is defective because it involves in part selective reporting, misuse of statistics, ignores evidence regarding the genetic/constitutional influences, and fails to consider published evidence regarding strong statistical relationships between air pollutant chemical concentrations and cancer mortality rates. It is also a fact that, despite many attempts, lung cancer has never been induced in experimental animals in well-designed studies by fresh tobacco smoke when experimental conditions resembled those of natural exposure.

As mentioned above, deficiencies in the theory that smoking causes lung and other cancers were examined at length by Burch in 1978. In 1965, K.A. Brownlee ["A Review of 'Smoking and Health'," Journal of the American Statistical Association 60, 722-739 (1965)] examined the 1964 Surgeon General's report, Smoking and Health, and found it defective in biochemistry and statistics. It is noteworthy that reports claiming that the smoking-mortality associations are causal appear largely in medical and biomedical journals rather than in statistical journals. In contrast, reports in statistical journals do not accept as valid the inference of causality from statistical association. It is also noteworthy that Brownlee's review was not mentioned in several subsequent Surgeon Generals' reports.

Finding (3). S. 1929 states that "heart disease accounts for nearly one-half of the deaths in the United States and one-third of the deaths attributable to heart disease are associated with smoking." Death rates from heart disease increase rapidly with increasing age, especially beyond 40 years. Thus, heart diseases appear to be in large part diseases of aging, a fact not evident in this finding.

The statement that one-third of these deaths are "attributable" to smoking implies cause. "Associated with" does not. There is no question about the association in some populations, but anyone who then argues for a causal relationship makes a huge leap based on faith alone. Any causal implication is defective because it involves in part those factors discussed earlier. In addition, quite strong statistical relationships between concentrations of a number of common air pollutant chemicals and mortality rates for several classifications of heart disease have been reported. The air pollution - heart disease relationship has often been overlooked or minimized in Surgeon Generals' reports.

Finding (4). S. 1929 states that "the risks of miscarriage, stillbirths, premature births, and child weight deficiencies for pregnant women who smoke are higher than for pregnant women who do not smoke." The term, "risks", is strictly statistical and is not biological or biochemical. The implication is that because of statistical associations, the smoking-causality explanation is true. This is the same misuse of statistics, and of science, noted previously.

Causality cannot be inferred from statistical association. The causal implications or claims, as stated in the Surgeon Generals' reports, are also defective because of selective reporting. Among the published reports that address the problem are: R.J. Hickey, R.C. Clelland, and E.J. Bowers, "Maternal Smoking, Birth Weight, Infant Death, and the Self-Selection Problem;" *American Journal of Obstetrics and Gynecology*, Vol. *131*, 805-811 (1978). Misuse of statistics is pointed out along with other errors and fallacies, including the overlooking of or subjective denigration of the publications of the late Professor J. Yerushalmy.

That low birth weight has been associated statistically with maternal smoking during pregnancy in some populations is not in question. However, the implications or claims in the Surgeon Generals' reports that the association means smoking-causality constitute misuse of statistics. Factors other than smoking also correlate with low birth weight, such as certain socio-economic variables, maternal height and weight, geographic differences, and altitude variations. The 2,500 gram birth weight criterion, implying that less than this weight is unhealthy, ignores the large variations in these factors. For example, there is evidence that birth weights of large, tall women tend to be heavier than birth weights of children of small, shorter women. [See J.M. Tanner, L. Lejarraga, and G. Turner, "Within-Family Standards for Birth-Weight," Lancet 2, 193-197 (1972)]. Surely constitutional factors influence birth size and weight in many populations.

An informative but controversial kind of evidence that addresses the smoking--causality versus the constitutional hypothesis pertains in part to whether women who later take up smoking ("future smokers") tended to have low birth weight children even before they started smoking. This question was examined by Professor J. Yerushalmy ["Infants with Low Birth Weight Born Before Their Mothers Started Smoking Cigarettes," American Journal of Obstetrics and Gynecology, Vol. 112, 277-284 (1972)]. He found that, indeed, birth weights of children of future smokers tended to be lower than birth weights of children of never smokers. Such a finding is in accord with the constitutional hypothesis rather than with the smoking-causality hypothesis. Yerushalmy's findings were consistent with those reported by D.T. Silverman [Maternal Smoking and Birth Weight, Thesis, Johns

Hopkins University, Baltimore, 1972]. These findings have not been widely popular.

It is also noteworthy that Yerushalmy found that the smaller babies of smoking mothers were healthier than the smaller babies of non-smoking women.

To summarize, in view of selective reporting in the Surgeon Generals' reports, the evidence supporting the constitutional hypothesis, the misused statistics and the sometimes biased samples, it seems desirable to table any legislative actions until the Congress has received a more complete analysis of the evidence. At the present time, statements about deleterious effects of smoking are often put forward supported by evidence that is scientifically unconvincing and possibly invalid.

Richard J. Hickey

Richard J. Hickey

March 2, 1982.

February 8, 1982

Biographical Sketch: Richard J. Hickey

Name: Richard James Hickey

Home Address: 43 E. Clearfield Road,
Havertown, Pa. 19083.

Business Address:
Department of Statistics,
The Wharton School,
University of Pennsylvania,
Philadelphia, Pa. 19104.

Birth Date: September 18, 1913.

Birth Place: Rock Island, Ill.

Marital Status:
Married, four children.

Telephone:
Home: (215) SU-9-8356
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Major Areas of Research Interest:

Epidemiological relationships between environmental chemicals and other environmental variables and various life forms, particularly man; natural selection; ecological genetics; mutagenesis; genetic polymorphism; human ecology; ecological statistical analysis; analysis of highly complex biological systems, such as multi-regional, multi-chemical epidemiological systems that pertain to elucidation of etiologies of major human afflictions, such as cancer, cardiovascular diseases, and birth defects; environmental pollution and public health; theoretical biology; behavior genetics; ethology, the biology of behavior; the molecular biological basis of biological senescence and of the development of diseases of aging; scientific methodology, valid and invalid; social consequences of abuse and misuse of statistics and of science generally. Also hormesis.

Education, College Level:

University of Illinois, Urbana, 1931-1935, B.S. in chemistry; mathematics minor.

University of Buffalo (N.Y.)(now S.U.N.Y.), graduate study in physical chemistry and chemical physics, 1936-1936.

Iowa State University, Ames, 1936-1941, Ph.D., biophysical chemistry, minor in microbiology.

Honorary Societies:

Sigma Xi, Pi Mu Epsilon, Phi Lambda Upsilon, Phi Kappa Phi.

Professional Organizations:

American Chemical Society; American Association for the Advancement of Science (AAAS Fellow); New York Academy of Sciences; Environmental Mutagen Society.

Teaching Experience:

Taught undergraduate chemistry at the University of Buffalo and at Iowa State University.

Professional Experience:

September, 1975 to present: Department of Statistics, The Wharton School, University of Pennsylvania, Philadelphia, Pa. Technical area as for August, 1973, to September, 1975.

August 1973 to September 1975: Management and Behavioral Science Center, Wharton School, University of Pennsylvania, Philadelphia, Pennsylvania; relationships of environmental chemicals and other variables to chronic disease risk; human ecology; population and molecular biology; theoretical biology; biology of behavior; complex biological systems analysis; multivariate analysis (applied); psychobiology and bioenergetics.

September 1967 to August 1971: Institute of Environmental Studies, University of Pennsylvania; also associated with Fels Center of Government, Wharton School, University of Pennsylvania, and with the Regional Science Research Institute, Philadelphia, Pa.; environmental pollution and chronic disease relationships; human ecology; multivariate analysis; complex biological systems analysis; theoretical biology.

1963 to September 1967: University of Pennsylvania, Institute for Cooperative Research, senior research investigator; much of these studies concerned national security and were classified. Studies included technical evaluations and feasibility, and multivariate analysis of complex systems-computer oriented. Also worked on the air pollution-public health program as a complex systems analysis problem in human ecology with a preventive medicine orientation.

1958 to 1963: Research and Development Department, Commercial Solvents Corporation, Terre Haute, Indiana. Planning and project analysis for Research and Development. Technical evaluations, feasibility studies, some market analysis and economic assessments. Work involved antibiotics, vitamins and other industrial biochemicals.

1953 to 1958: University of Pennsylvania, Institute for Cooperative Research, senior research investigator. Work was somewhat similar to that of 1963-1967. Worked on computer-oriented predictive models of various sorts. Some work was published in scientific journals.

1941 to 1953: Similar to work of 1958-1963, but more laboratory work. Was involved in research and development work for the first penicillin manufacturing plant ever built.

Recent Abstracted Descriptions of Current Studies.

Item 803: "Ecological Statistical, Ethological, and Other Biological Studies on Air Pollution, Smoking, and Other Environmental Variables and Their Relation to Chronic Diseases in Man," pp. 326-327, in: 1976 Directory of On-Going Research in Smoking and Health, U.S. Department of Health, Education and Welfare, PHS, Center for Disease Control, National Clearinghouse for Smoking and Health, Atlanta, Ga.

Item 834/12.76: "Ecological Statistical, Ethological, and Related Biological Studies on Air Pollution, Smoking, and Other Environmental Variables and Their Relation to Chronic Diseases in Man," pp. 384-385, in: Directory of On-Going Research in Cancer Epidemiology, 1977; International Agency for Research on Cancer (IARC), Lyon, France; Deutsches Krebsforschungszentrum (DKFZ), Heidelberg, West Germany. (IARC Scientific Publ. No. 17).

List of Publications

Industrial Microbiology and Biochemistry

1. Shriner, R. L., and Hickey, R. J., Urethans as local anesthetics (V), alkyl ω-diethylaminopropylcarbamates, *J. Am. Chem. Soc. 61*, 888-9 (1939); correction, *J. Am. Chem. Soc. 63*, 3549 (1941).

2. Fulmer, E. I., Hickey, R. J., and Underkofler, L. A., Determination of glycerol by oxidation with ceric sulfate in fermentation media containing dextrose, *Ind. Eng. Chem., Anal. Ed. 12*, 729-30 (1940).

3. Hickey, R. J., Sterility test for penicillin employing cysteine for inactivation, *Science*, *101*, 232-4 (1945).

4. Hickey, R. J., In vitro evidence of regeneration of active penicillin from penicillin esters, *Science*, *101*, 462-3 (1945).

5. Hickey, R. J., The inactivation of iron by 2,2'-bipyridine and its effect on riboflavin synthesis by *Clostridium acetobutylicum*, *Arch. Biochem. 8*, 439-47 (1945).

6. Fulmer, E. I., Underkofler, L. A. and Hickey, R. J., Glycerol production, U. S. Patent 2,388,840 (1945).

7. Gilbert, W. J., and Hickey, R. J., Production of conidia in submerged cultures of *Penicillium notatum*, *J. Bact. 51*, 731-3 (1946).

8. Hickey, R. J., Precipitation of riboflavin from aqueous solution by bacteriological reduction, *Arch. Biochem. 11*, 259-67 (1946).

9. Hickey, R. J., Production of riboflavin by butyl alcohol-producing bacteria, U. S. Patent 2,425,280 (1947).

10. Fulmer, E. I., Underkofler, L. A., and Hickey, R. J., Glycerol production by fermentation process, U. S. Patent 2,416,745 (1947).

11. Hickey, R. J., A note on detergent interference in the serial dilution assay of penicillin using *Bacillus subtilis*, *J. Bact. 55*, 43-4 (1948).

12. Hickey, R. J., and Hidy, P. H., Crystalline fradicin, *Science 113*, 361-2 (1951).

13. Hidy, P. H., and Hickey, R. J., The isolation of cystalline fradicin from fermentation media, *Arch. Biochem. Biophys. 34*, 67-71 (1951).

14. Underkofler, L. A., Fulmer, E. I., Hickey, R. J., and Lees, T. M., Production of glycerol by fermentation. I. Fermentation of dextrose, *Iowa State Coll. J. Sci. 26*, 111-33 (1951).

15. Hickey, R. J., Oral therapeutic tablets (Penicillin), U.S. Patent 2,566,200 (1951).

16. Hickey, R. J., Production of riboflavin, U.S. Patent 2,605,210 (1952).

17. Hickey, R. J., Corum, C. J., Hidy, P. H., Cohen, I. R., Nager, U.F.B., and Kropp, E., Ascosin, an antifungal antibiotic produced by a streptomycete, *Antibiotics and Chemotherapy* *2*, 472-83 (1952).

18. Hickey, R. J., and Tresner, H. D., A cobalt-containing medium for sporulation of *Streptomyces* species, *J. Bact.* *64*, 841-2 (1952).

19. Hickey, R. J., Production of penicillin, U.S. Patent 2,615,830 (1952).

20. Hickey, R. J., Some nutritional requirements for biosynthesis of riboflavin by *Eremothecium ashbyii*, *J. Bact.*, *66*, 27-33 (1953).

21. Hickey, R. J., The antagonism between the antifungal antibiotic, ascosin, and some long-chain, unsaturated fatty acids, *Arch. Biochem. Biophys.* *46*, 331-6 (1953).

22. Underkofler, L. A., and Hickey, R. J. (editors), "Industrial Fermentations", Vol. I, (book), Chemical Publishing Co., New York, 1954. Includes a chapter by W.H. McCutchan and R. J. Hickey on "The butanol-acetone fermentation."

23. Underkofler, L. A., and Hickey, R. J. (editors), "Industrial Fermentations", Vol. II (book), Chemical Publishing Co., New York, 1954. Includes a chapter by R. J. Hickey on "Production of riboflavin by fermentation," and a chapter by R.J. Hickey and A. R. Stanley on "Miscellaneous fermentations."

24. Hickey, R. J., and Phillips, W. F., Separation of Aureomycin from Terramycin by countercurrent distribution and by paper chromatography. *Anal. Chem.* *26*, 1640-2 (1954).

25. Hickey, R. J., Soluble ascosin composition and method for preparing same, U.S. Patent 2,748,053 (1956).

26. Hickey, R. J., Production of riboflavin by *Ashbya gossypii* with cobalt as additive, U.S. Patent 2,667,445 (1954).

27. Hickey, R. J., Survey of fermentations. This was a paper presented at a symposium on Non-food Uses of Carbohydrates at the American Chemical Society convention, Atlantic City, September, 1959.

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COLLEGE OF MEDICINE AND DENTISTRY OF NEW JERSEY

NEW JERSEY MEDICAL SCHOOL

100 Bergen Street
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Statement

of

Duncan Hutcheon, M.D., D.Phil.

Departments of Pharmacology and Medicine

March 12, 1982

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My name is Duncan Hutcheon. I am Professor of Pharmacology and Medicine at the College of Medicine and Dentistry of New Jersey. I am also an attending physician at the College Hospital in Newark and am director of the Clinical Pharmacology Training Program at the New Jersey Medical School, where my responsibilities have included serving on the Human Studies Research Committee. I am a graduate of the University of Toronto where I received my M.D. and B.Sc.(Med.) degrees. In 1950, as a National Research Council (Canada) Postdoctoral Fellow, I obtained my Ph.D. at Oxford University in the Department of Pharmacology.

My research has been primarily in the field of cardiovascular and clinical pharmacology, and I have approximately 63 research publications. In addition, I have contributed sections of several textbooks, including Drill's Pharmacology and Medicine, Treatment of Heart Disease in the Adult by Rubin, et al., and Cardiovascular Therapy by Russek. Since 1977, I have served as editor of the Journal of Clinical Pharmacology.

I hold memberships in numerous medical and scientific societies including the American Society for Pharmacology and Experimental Therapeutics and the Society for Experimental Biology and Medicine. I am a fellow of the American College of

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Physicians and the American College of Clinical Pharmacology of which I am a past-president.

In May, 1980, I helped establish the Princeton Institute of Environmental Medicine (PIEM), an organization of medical scientists concerned about the health effects of chemical substances in the environment. Our research focuses primarily on the relationship between various environmental risk factors and chronic diseases such as heart disease and cancer. The environmental factors we are investigating include tobacco smoke constituents, industrial chemicals, pesticides, and emissions from jet, diesel, and gasoline engines.

It is the consensus of those associated with the work of the Institute that it is impossible to make strong, unequivocal statements about the contribution of any single factor to the occurrence of cancer and heart disease. For this reason, many of the statements in H.R. 4957 and S. 1929 regarding the health and economic consequences of smoking are unacceptable scientifically and appear to represent an attempt to offer easy solutions to what are actually complex multifaceted problems.

Furthermore, it is our belief that Congress has the responsibility of encouraging thorough and unbiased investigation into the spectrum of risk factors associated with these diseases

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rather than limiting its consideration to only one aspect. Why, for example, doesn't the proposed legislation mention environmental hazards? After all, in terms of both quantity and inherent carcinogenicity and cardiotoxicity, environmental chemicals such as polycyclic hydrocarbons (PAH's), halogenated hydrocarbon pesticides and industrial solvents appear to pose serious public health problems.

These views are based on our field work that involves correlating mortality data provided by the National Institutes of Health with our own clinical and laboratory findings. By this method, we have examined the following environmental pollutants: benzo(a)pyrene (BaP) and other PAH's, carbon monoxide (CO), and halogenated hydrocarbon pesticides and industrial solvents.

Our studies show that there is considerable misinformation regarding the possible biologic effects of the various chemicals to which we are regularly exposed. Moreover, there is little appreciation of the way in which such substances are handled by the body (i.e., how and when they are eliminated), and the relationship between the amounts of chemicals present in the environment and the amounts necessary to initiate carcinogenic and cardiotoxic activity.

For example, we measured serum B(a)P levels by radioimmunoassay procedure in urban and suburban population groups in the New York metropolitan area and found significantly higher levels in the group living in the areas with highest atmospheric pollution. B(a)P was chosen as a marker of urban air pollution because it is a suspected carcinogen that is produced by burning fossil fuels. It is also considered to be a valid indicator of potentially carcinogenic PAH's in the atmosphere. Our observations indicate that geographic location may be an important risk factor for respiratory cancer. While these results represent only preliminary findings, they suggest that environmental studies should be conducted on a much larger scale. Such studies could assist in identifying populations at environmental risk and pinpointing geographic areas where environmental control efforts should be directed.

During the past three years, my laboratory has also focused on the effects of environmental chemicals on cardiac performance. This has involved investigating the association between geographic location and the prevalence of heart disease and sudden cardiac death. Our findings suggest that certain chemicals associated with adverse cardiologic effects are present both in the ambient air over Newark and in the drinking water supply of suburban communities. The chemical agents that may be related to cardiac mortality rates in areas of greatest risk are the halogenated hydrocarbons.

Although we originally assumed that CO might play some role in heart disease causation, our findings have suggested that (1) CO does not appear to predispose the heart to catecholamine-induced arrhythmias in experimental animals; and (2) although the CO levels are higher in the ambient air in the urban-industrial section of Newark than in the New York suburbs, the concentrations do not seem high enough to cause health consequences.

Our laboratory studies have also led us to an appreciation of the importance of the absorption/elimination characteristics of environmental chemicals. It is our view that the toxic properties of foreign chemicals ultimately depend on how well the body biotransforms and eliminates them and on how the body's immunological and other defense systems operate. It is clearly evident that more research is needed on the health effects of long-term, low level exposure to chemicals.

In summary, our work at PIEM indicates that a balanced scientific approach is necessary in the study of the health consequences of environmental risk factors. Any approach that limits its consideration to only one factor, like tobacco smoke, will hinder the generation and analysis of the epidemiological, pharmacokinetic, and toxicologic data needed to effectively study the causes of our nation's health problems.

STATEMENT OF LEON O. JACOBSON

I am Leon Orris Jacobson from The University of Chicago in Chicago where I continue as a physician-scientist emeritus and have served as Chairman of Medicine, Dean of Medicine and Biology and Director of the Argonne Cancer Research Hospital. In February 1942 I became responsible for the medical safety of the personnel of the Metallurgical Laboratory (the atomic bomb project) under the direction of Arthur Compton, Norman Hillberry, Enrico Fermi, Leon Szilard and many others who achieved the first self-sustaining chain reaction. Later I served as Associate Director and then Director of Medicine and biology of this enormously successful national program that brought the war abruptly to an end and in the aftermath provided the technology and the radioisotope tracers so enormously important to the revolutionary advances in biology and medicine that contribute an ever increasing momentum to our understanding of normal as well as disease processes.

I joined the Scientific Advisory Board of the Tobacco Industry Research Committee (now called the Council for Tobacco Research - U.S.A., Inc.) in 1954 when it was under the leadership of Clarence Cook Little. Dr. Little appeared before Congressional committees dealing with smoking and health in 1965 and 1969. The mandate of the Scientific Advisory Board was broad and simple, namely to sponsor independent research in the area of tobacco and health. The Scientific Advisory Board was and is today composed of individual scientists whose expertise covers those disciplines of the biomedical sciences that are essential to understanding the complexities of chronic diseases and their pathogenesis.

In spite of the successful elimination of many diseases caused by infectious agents, we are still groping for an understanding of the inexorable process of chronic diseases, such as arteriosclerosis and cancer. Are we in any better position today to fathom the challenging puzzle of cause and cure of these devastating medical problems?

We are becoming more aware of the fact that cancer, arteriosclerosis, and other chronic diseases are not the result of exposure to a single substance that has been inhaled or eaten. For example, arteriosclerosis is hastened by a concomitant hypertension, but occurs even in people who are of normal weight and without hypertension. What causes the arteriosclerosis that leads to coronary occlusions and strokes? Here we have a controversy that will be resolved only when each of the categories of research unravels the cascade of events that leads to death or disability. It would be disastrous, for example, to decide now that arteriosclerosis is primarily a problem relating to blood lipid control.

Cancer etiology and control have been intensively researched, especially in the past 75 years. We have learned much from the rapidly developing area of molecular biology which is driving us nearer and nearer to the ultimate goal of understanding the thousand and one continuous reactions in cell membranes, within cells and in organs and tissues that are required for maintaining the normal steady state. We are just beginning to understand the interrelationships of individual\ cells, tissues and organs such as the lung, kidney, and adrenals as well as the brain itself. Each of these body constituents is not an independently functioning entity, but rather part of a wonderful organism working in harmony in the healthy individual which quite often reverses or corrects abnormalities (or copying mistakes) that result spontanously or from injury or disease. Spontaneous mistakes in the process of cell division are inevitable because millions of cells must yield exact copies of the genetic information they possess to their progeny. The body has the ability to repair the strands of genetic material in which a mistake occurred in the reproduction process. If the repair is incomplete or fails then the newly formed cell might die or it may go on to develop a new kind of cell - a cancer cell.

How else might cancer occur? We know that selected mice can be inbred in a deliberate [illegible] (by 20 generation crosses) and a predictable percentage of their progeny will develop cancer. This is a very useful laboratory technique for testing carcinogenicity or cocarcinogenicity of a given chemical or other agent. Likewise, epidemiologists have found and are closely observing human "cancer families" in which the genetic role is clear. Thus, a full understanding of the complex process of carcinogenesis must consider not only abnormal inherited genes or chemical exposure of the genetic material of a cell but also the possible impact, involvement or participation of ionizing radiations and ultraviolet rays, viruses and many internal derangements.

In the laboratory animal, when studying tumor induction with a given chemical substance or virus, one has better control of the various factors which may play a role in carcinogenesis, but in human epidemiological studies it is very difficult to isolate one factor from another. Human beings are exposed to a variety of environmental impingements. They also have differing life styles involving various stresses, eating habits, drinking or smoking habits, as well as different genetic or constitutional backgrounds. We are seeking ways and there are promising leads in the search for the members of our population who may be susceptible or likely to develop cancer. We have found families with genetically transmitted susceptibility to emphysema. Likewise in the field of cancer, we are now looking for markers to identify people who might be susceptible.

Why have I stressed the importance of spontaneous, inherited or induced genetic aberration in the development of disease? Simply because many of our dread diseases seem to originate after a lesion occurs in a DNA strand, which may actually lead to the development of an individual cancer or become an inherited problem in successive generations. For example, molecular biology has already located the particular sequence in the double helix responsible for sickle cell anemia. Genetic engineering is a thing of the present that promises rapid progress.

Malignant cells carry unique and identifying molecules on their surfaces that are characteristic of the specific malignancy. With current techniques immunologists can develop Killer T lymphocytes that recognize and seek out the specific molecules of malignant cells and destroy the malignancy by attacking the surface molecules of the target malignant cells.

Many have heard of the hybridoma - a powerful tool that is revolutionizing biology. In the test tube a fusion of mouse tumor cells and antibody-forming cells results in a hybrid cell that can generate, perhaps immortally, large quantities of antibodies that will react with small peptide chains that are a part of a large protein. These hybridoma antibodies may also be used to treat tumors. This is an example of what genetic engineering can and is doing. We shall see in the next decade, and, in fact, we are already witness to a wide spectrum of genetic engineering procedures.

Even as I stand here at age 70, I am hopeful that we shall see the answers before the end of this century to many of the diseases that plague us. We are in the middle of vast and fruitful fields of molecular genetics. We can find the genetic abnormalities and we can even splice in new gene segments. We are literally swimming in new knowledge in immunology and only beginning to apply the fruits of these discoveries.

This has been a remarkable era of discovery in the area of peptide hormones which exert tremendous influence on many functions, including lung and cardiovascular. The future challenge is to see whether, and how, these peptides may play a role in the causation of chronic disease.

From these continuously emerging discoveries, only a few of which I have touched upon today, the synthesis of disparate findings occurs. It is this synthesis which heralds the understanding of the aging process. Only such understanding will lead to control of our most formidable and complicated diseases such as arteriosclerosis, heart diseases and cancer, as well as the whole category of other degenerative maladies.

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Born December 16, 1911, in Sims, North Dakota

Married; two children, Eric and Judith

Degrees: B.S., 1935, North Dakota State University

M.D., 1939, The University of Chicago

Honorary Degree of Doctor of Science, 1966, North Dakota State University

Honorary Degree of Doctor of Science, 1972, Acadia University, Nova Scotia

Licensure and Certification:

State of Illinois

American Board of Internal Medicine

Academic Positions:

The University of Chicago

Intern, 1939-40

Assistant Resident in Medicine, 1940-41

Assistant in Medicine, 1941-42

Instructor in Medicine, 1942-45

Assistant Professor of Medicine, 1945-48

Associate Dean, Division of the Biological Sciences, 1945-51

Associate Professor of Medicine, 1948-51

Professor of Medicine and Head of the Hematology Section, 1951-61

Professor and Chairman, Department of Medicine, 1961-65

Joseph Regenstein Professor of Biological and Medical Sciences, 1965- 81 Emeritus 1981 -

Dean, Division of the Biological Sciences and the Pritzker School of Medicine, 1966-1975

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Special Appointments:

The University of Chicago

Health Officer, Metallurgical Laboratory (Plutonium Project), Manhattan District, Medicine and Biology, 1942-43

Associate Director of Health, Metallurgical Laboratory (Plutonium Project), Manhattan District, 1943-45

Director of Health, Metallurgical Laboratory (Plutonium Project), Manhattan District, 1945-46

Member, Institute of Radiobiology and Biophysics, 1949-54

Director, Argonne Cancer Research Hospital (operated by The University of Chicago for the U.S. Atomic Energy Commission), 1951-67

Director, Franklin McLean Memorial Research Institute (operated by The University of Chicago for the U.S. Atomic Energy Commission/Energy Research and Development Administration), 1974-77

Consultant:

Consultant, Division of Biological and Medical Research, Argonne National Laboratory

Special Consultant, [illegible]atology Study Section, U.S. Public Health Service, 1949-[illegible]

Consultant, Division of Radiobiological Health, University Grant and Training Branch, Department of Health, Education, and Welfare, 1962-65

Consultant, University of California, Berkeley, 1962-

[illegible]

Boards and Committees:

Member, Committee for Radiation Studies, U.S. Public Health Service, 1951-55

Member, Committee on Cancer Diagnosis and Therapy, Panel on Chemotherapy, National Research Council, 1951-55

Member, Advisory Committee on Isotope Distribution, U.S. Atomic Energy Commission, 1952-56

United States Representative, First and Second International Conferences on Peaceful Uses of Atomic Energy, Geneva, 1955 and 1958

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Boards and Committees (cont'd):

Member, National Advisory Board of the Oklahoma Medical Research Foundation, 1959-64

Member of Advisory Committee sent to Yugoslavia, U.S. Atomic Energy Commission and the Department of State, 1960

Member, Expert Advisory Panel on Radiation, World Health Organization, Geneva, 1960-65

Member, Advisory Committee on Biophysics, Surgeon General of the Army, 1961-70

Member, Morison Panel of the Wooldridge Committee, Office of Science and Technology, Executive Office of the President, 1963-65

Member, Medical and Scientific Advisory Board, Will Rogers Memorial Fund, 1963-66

Member, Erythropoietin Committee, National Heart Institute, U.S. Public Health Service, 1963-67

Member, Board of Scientific Counselors, National Cancer Institute, National Institutes of Health. 1963-67

Member, Space Biology Advisory Subcommittee, Space Science and Applications Steering Committee, National Aeronautics and Space Administration, 1968-70

Member, National Advisory Cancer Council and National Cancer Advisory Board, National Institutes of Health, 1968-72

Member, Subcommittee of the Mayor's Search Committee for Commissioner of Health, Chicago, 1969

Member, Medical Advisory Committee, Civil Service Commission of Cook County, 1969

Member, Environmental Advisory Council, Commonwealth Edison Company, Chicago, 1970

Member, International Board of Editors, *Excerpta Medica,* The Netherlands, 1970-81

Member, Editorial Board, *Contemporary* Medicine and Surgery McGraw-Hill, Inc.

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Boards and Committees (cont'd):

Member, President's Advisory Committee on the Puerto Rico Nuclear Center, University of Puerto Rico, San Juan, 1970-

Chairman, Committee on the Study of Postdoctoral Fellowship and Traineeship Programs in the Biomedical Sciences, National Academy of Sciences-National Research Council, 1971-72

Member, Report Review Committee, National Academy of Sciences, 1971-72

[illegible]

Member, Illinois Institute of Health and Public Health Advisory Committee, 1969

Member, Board of Directors; member, Regional Advisory Group, Illinois Regional Medical Program, 1971-73

Member, Council of Medical Deans, State of Illinois 1964-74

Member, Dean's Advisory Committee for Education in the Health Fields, State of Illinois

Member, Public Health Advisory Committee, State of Illinois, Board of Higher Education

Member, Advisory Committee on Personnel for Research, American Cancer Society

Member, Scientific Advisory Board, Institute for Advanced Learning in the Medical Sciences, City of Hope Medical Center, California, 1962-

Member, Board of Trustees, La Rabida Children's Hospital and Research Center, Chicago, 1967-

Director at Large, Board of Directors, Alpha Omega Alpha Honor Medical Society, 1967-71

Member, Committee on Honorary Memberships, Alpha Omega Alpha Honor Medical Society, 1971-72

[illegible]

Member, Liaison Subcommittee of the Joint Committee on Tobacco and Health, Department of Health, Education, and Welfare

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Boards and Committees (cont'd):

Member, Medical Advisory Board, Leukemia Research Foundation

Member, Scientific Advisory Committee, Institute for Cancer Research, Philadelphia, 1959- 72

Member, Scientific Advisory Board, The Council for Tobacco Research - USA, New York 1954 - Chairman, 1981 -

Member, Steering and Finance Committee, American Society of Hematology, 1973

Member, Committee on Professional Education, The Institute of Medicine of Chicago, 1967-

Member, Evaluation Committee, National Fund for Medical Education, 1966-

Member, Subcommittee on Hematopoietic and Lymphatic System of the Committee on Rating of Mental and Physical Impairment, American Medical Association, 1967

Chairman, Committee on Science Policy for Medicine and Health, Institute of Medicine, National Academy of Sciences, 1972-73

Member, Board of Governors, The American Society of Contemporary Medicine and Surgery 1966

Chairman, Section of Medical Sciences, National Academy of Sciences, 1974- 77

Member, Committee on Federal Research on Biological and Health Effects of Ionizing Radiation (FREIR), National Research Council, 1979 - 81

Lecturer: Medical Aspects of Atomic Warfare, Atomic Energy Commission Training Program, Chicago, 1950

Fifth International Cancer Congress, Paris, 1950

International Congress of Radiology, London, 1950; Munich, 1959

International Society of Hematology, University of Cambridge, England, 1950; Munich, 1959

American College of Physicians, University of Chicago, 1950

Symposium on Radiology, Cleveland, 1950

Chicago Society of Internal Medicine, Chicago, 1951

Chicago Pathological Society, Chicago, 1951

Northern Indiana Medical Society, Fort Wayne, 1951

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Lecturer (cont'd):

Utah Medical School, Salt Lake City, 1951

Rheumatic Fever Research Institute, Chicago, 1951

"Know Your Chicago," course sponsored by University College, University of Chicago, at the Art Institute, Chicago, 1951

Chicago Cancer Research group, Billings Hospital, Chicago, 1951

Hematology Club, Chicago, 1951

Naval Medical Research Institute, sponsored by American Institute of Biological Sciences, Bethesda, 1951

Nursing Service Department, Chicago Chapter of American Red Cross, 1951

Congresses of the International Society of Haematology: Argentina, 1952; Paris, 1954; Boston, 1956; Rome, 1958; Tokyo, 1960; Mexico City, 1962; Stockholm, 1964; Munich, 1970

R. W. Stewart Memorial Lecture, Pittsburgh Academy of Medicine, 1952

Janeway Lecture, American Radium Society, 1953

CIBA Foundation, London: Conference on Experimental Leukaemias, 1953; Symposium on Haemopoiesis, 1960

Symposium on Radiobiology, Liege, 1954

Burrell O. Ralston Lecture, University of Southern California, 1957

Alice Messinger Band Memorial Lecture in Hematology, University of Maryland, 1959

George Minot Memorial Lecture, American Medical Association, 1960

Jacobaeus Memorial Lecture for 1962, Helsinki

Malthe Foundation Lecture for 1962, Oslo

Guest Lecturer, Argentine Cancer Society and Liga Argentina de Lucha contra el Cancer, Buenos Aires, 1963

Kennecott Lecture Series, University of Arizona, 1963

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Lecturer (cont'd):

Kretschmer Memorial Lecture, Institute of Medicine of Chicago-Chicago Society of Internal Medicine, 1968

Medical Sciences Symposium, Abbott-Northwestern Hospital, Minneapolis, 1970

Symposium Erythropoieticum, XVth Scientific Conference of the Faculty of General Medicine, Charles University, Prague, 1970

Symposium, "Radiobiology, Past and Present," to honor Austin M. Brues, Argonne National Laboratory, 1971

11th Annual Dwight E. Clark Lecture, Roane Anderson Medical Society, Oak Ridge National Laboratory, 1971

Conference on Planning for Cancer Centers, sponsored by American Cancer Society and National Cancer Institute in cooperation with Association of American Medical Colleges, Washington, D.C., 1971

Special Session on the History of Bone Marrow Transplantation, Experimental Hematology Society, Davis, California, 1971

Scientific Session, The Installation of Dr. Charles Brenton-Huggins as Chancellor of Acadia University, Wolfville, Nova Scotia, March, 1972

Honors and Awards:

Member, Alpha Omega Alpha Honor Medical Society

Member, Sigma Xi

Janeway Medal, American Radium Society, 1953

Robert Roesler de Villiers Award, Leukemia Society, 1956

Ralston Award, University of Southern California, 1957

Borden Award for research in the medical sciences, Association of American Medical Colleges, 1962

Elected Member, Academia Nacional de Medicina de Buenos Aires, 1963

Modern Medicine Award for distinguished achievement, 1963

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Honors and Awards (cont'd):

American Nuclear Society Award for distinguished service in the advancement of nuclear sciences, 1963

Elected Member, National Academy of Sciences, 1965

City of Hope National Medical Center Citation for distinguished leadership in medical education and research, 1967

Elected Fellow, American Academy of Arts and Sciences, 1967

Elected Master, American College of Physicians, 1968

The John Phillips Memorial Award, American College of Physicians, in recognition of distinguished achievement in Internal Medicine,

Theodore Roosevelt Rough Riders Award, State of North Dakota, 19

Elected a Laureate of the Lincoln Academy of Illinois and recipient of Order of Lincoln in Medicine and Health, 1979

Societies:

Academia Nacional de Medicina de Buenos Aires

Alpha Omega Alpha Honor Medical Society

American Academy of Arts and Sciences

American Association for Cancer Research

American Association for the Advancement of Science

American College of Physicians

American Medical Association

American Nuclear Society

American Society for Clinical Investigation

American Society for Experimental Pathology

American Society of Hematology

Association of American Physicians

Billings Medical Club

Central Interurban Club

Central Society for Clinical Research

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Journal of the American Medical Association, 117: 2222-2225, 1941.

3. Jacobson, Leon Orris, and Palmer, Walter Lincoln: The differential diagnosis of duodenal ulcer.
Medical Clinics of North America, 27: 195-207, 1943.

4. Jacobson, Leon O., and Palmer, Walter Lincoln: The effect of anemia on gastric emptying.
Gastroenterology, 1: 1133-1140, 1943.

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Endocrinology, 34: 240-244, 1944.

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Journal of the American Medical Association, 132: 263-271, 1946.

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Proceedings, Central Society for Clinical Research, 19: 30, 1946.
Journal of Laboratory and Clinical Medicine, 32: 341, 1947.

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Radiobiology, 49: 286-298, 1947.

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In *Approaches to Tumor Chemotherapy* (1st Ed.), Forest Ray Moulton (ed.), Lancaster Press Printing Co., Lancaster, Mass., Chap. V, pp. 306-318, 1947.

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American Journal of Roentgenology and Radium Therapy, 58: 774-782, 1947.

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Chicago Medical Society Bulletin (North Shore), 3: 1947.

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Anatomical Record, 99: 576, 1947.

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Radiology, 50: 387-394, 1948.

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Science, 107: 248-250, 1948.

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Anatomical Record, 100: 46, 1948.

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American Journal of Clinical Pathology, 18: 671-689, 1948.

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Proceedings of the Society for Experimental Biology and Medicine, 69: 84-86, 1948.

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Journal of Laboratory and Clinical Medicine, 33: 1649, 1948.

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Journal of Laboratory and Clinical Medicine, 33: 1480, 1948.

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Proceedings of the Society for Experimental Biology and Medicine, 69: 548-554, 1948.

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Journal of the American Medical Association, 139: 138-140, 1949.

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The American Journal of Medical Technology, 15: 86-87, 1949

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Journal of Clinical Investigation, 28: 465-468, 1949.

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Journal of Laboratory and Clinical Medicine, 34: 902-924, 1949.

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Journal of Laboratory and Clinical Medicine, 34: 1366-1375, 1949.

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Journal of Laboratory and Clinical Medicine, 35: 252-264, 1950.

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Journal of Laboratory and Clinical Medicine, 36: 800, 1950.
Proceedings, Central Society for Clinical Research, 23: 12, 1950.

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Medical Clinics of North America, 35: 93-110, 1951.

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Science, 113: 510-511, 1951.

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Proceedings of the Institute of Medicine of Chicago, 18: 326, 1951.

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Proceedings of the Chicago Pathological Society, 13: 84-85, 1952.

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Journal of Laboratory and Clinical Medicine, *41*: 499-515, 1953.

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Journal of the American Medical Association, *152*: 1018-1028, 1953.

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Proceedings of the Society for Experimental Biology and Medicine, *85*: 682-685, 1954.

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Proceedings of the Society Experimental Biology and Medicine, 92: 203-207, 1956.

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STATEMENT OF LAWRENCE L. KUPPER, Ph.D.

1. Introduction

I am Lawrence L. Kupper, Ph.D., a biostatistician specializing in epidemiology and environmental health, currently Professor of Biostatistics in the School of Public Health, University of North Carolina at Chapel Hill. I have been a consultant to the U.S. Environmental Protection Agency and a member of the Instructional Staff of that Agency. I am currently doing research under a grant from the National Institute of Environmental Health Sciences.

Since 1967, I have been engaged in biostatistical research with particular reference to the analysis of epidemiological data on occupational health and cancer. I am an elected member of the Regional Committee of the Eastern North American Region of the Biometric Society and am a member of the American Statistical Association and the Society for Epidemiologic Research. I have published or presented approximately 100 scientific papers and am on the Editorial Board of the Journal of Chronic Diseases. My curriculum vitae and list of publications are attached.

My appearance at this hearing is voluntary and the opinions expressed are personal, not representing those of any organization.

2. Discussion

The widely-held belief that smoking "causes" lung cancer stems mainly from associations encountered in a wide variety of epidemiologic studies on human populations. Accordingly, it is important to discuss some of the limitations inherent in the use of epidemiologic research studies to assess the purported smoking-lung cancer relationship.

First, epidemiologic studies are generally observational rather than experimental. Epidemiologists are not able to conduct controlled experiments in laboratory-type settings where environmental conditions can be strictly monitored. Epidemiologists generally have no control over what their experimental units (i.e., human subjects) may be exposed to over a lifetime. A human being is exposed to a myriad of environmental impingements over a lifetime; these impingements take many forms (e.g., air pollution, diet, stress, exposure to occupational hazards, etc.), and they vary both in intensity and in the sequence in which they occur. Furthermore, the biological mechanisms by which such agents may act, either separately or in combination, to produce an adverse health effect are generally unknown.

If we want to compare two groups of individuals (e.g., smokers and non-smokers) with regard to their rates of development over time of some disease (e.g., lung cancer), how can we best deal with the fact that study populations are different with regard to genetic background, environmental impingements and so forth? We would be fairly content if we knew that these two groups were, on the average, equally susceptible to the disease before the suspected causal agent is introduced, regardless of previous exposure histories with respect to all other environmental impingements. One way to try to achieve such a desirable starting place for a follow-up study would be to randomly assign individuals to one of the two groups. This process of randomization represents an attempt to insure comparability, on the average, between the two groups with respect to both measured and unmeasured factors which vary from individual to individual and which may be risk factors for the disease under study. Although randomization is by no means a 100% guarantee that exact comparability will be achieved, it is certainly one reasonable way to balance the distribution of such factors (e.g., previous unknown exposures, genetic differences, diet, psychological and sociological factors, etc.) between the two groups.

Unfortunately, the technique of randomization is not available to the epidemiologist in most situations. For example, people who smoke have chosen to smoke, and people who do not smoke have chosen not to smoke. Thus, any epidemiologic study comparing smokers and non-smokers suffers from the basic flaw that the individuals have self-selected themselves into these two distinct groups. Thus, any observed differences between two such groups with respect to health outcomes may not, in fact, be the result of smoking, but may instead be due to other more basic factors (e.g., those of a genetic origin) which are necessarily different between the two groups because of the self-selection process itself. In other words, one can reasonably maintain the view that smokers are constitutionally different from non-smokers, and that such constitutional factors cause such individuals both to smoke and to develop lung cancer. Under this plausible hypothesis, then, smoking is an outcome variable just like lung cancer, being a manifestation of the constitutional factors uniquely possessed by people who choose to smoke.

Actually, there is considerable evidence that smokers are, indeed, a constitutionally different group of people from non-smokers (Burch, 1978). For example, many studies have shown that smokers differ, on the average, from non-smokers with respect to morphology, personality, and genetic markers. Studies of twins have shown that pairs of monozygotic twins (whether brought up together or apart) have much more similar smoking habits than do pairs of like - sex dizygotic twins, thus suggesting a genetic link with the tendency to smoke.

With regard to the relationship between genetic differences among individuals and differing levels of disease susceptibility, Janis and Kupper (1982) have recently demonstrated by sophisticated statistical methodology, the existence (for U.S., and also for British, male lung cancer data) of a particular birth cohort (i.e., that group of individuals born around the end of the nineteenth century) which appears to be much more prone to develop lung cancer than do other birth cohorts born either before or after that time. The existence of such high-risk birth cohorts suggests that genetic, prenatal, and neonatal factors can be important determinants of subsequent disease susceptibility.

Based on all these findings, the self-selection bias problem is a very real one, which casts serious doubt on the ability of epidemiologic research studies to provide final answers regarding the causal relationship between smoking and lung cancer.

In addition, there are several other sources of bias that typically affect the validity of epidemiologic studies designed to address the smoking-lung cancer causality issue. These are:

1. Various studies (e.g., several of those referred to in the latest Surgeon General reports) involve particular sub-groups of individuals, which are in no way representative of the general population and which are excellent illustrations of the self-selection syndrome. Examples of such studies are the British male doctor study of Doll and Hill (1964), and the studies of Seventh Day Adventists (e.g., see Lemon and Walden, 1966) and Mormons (see Enstrom, 1975). It is well known that doctors differ from the average person in very many respects; and, members of religious sects which prohibit the use of alcohol and tobacco generally are much different personality-wise from the general population. Clearly, findings regarding such unique sub-groups cannot be applied to the general population.

2. It is well documented that the diagnosis of lung cancer is a difficult clinical problem and that, in fact, the diagnostic criteria and associated diagnostic error rates have changed considerably over the years. Such changes can undermine the validity of associations between smoking and lung cancer reported in various epidemiologic studies.

3. A more subtle form of bias, known as detection bias, has been discussed by Feinstein and Wells (1974), specifically with regard to the smoking-lung cancer controversy. Generally speaking, such a bias can arise when the knowledge of exposure or non-exposure to an agent alters the objectivity of the scientist. For example, an M.D. who knows a person is a heavy smoker may be more on the lookout for lung cancer in that person than he would be for a light smoker or a non-smoker. This form of detection bias would obviously tend to exaggerate the smoking-lung cancer association, as has been demonstrated by Feinstein and Wells with real-life data.

4. It is well-known that there is considerable inaccuracy associated with the use of exposure information based on questionnaires and interviews with next-of-kin to assess

habits of deceased relatives with regard to smoking or drinking, even though this is often the only source of such information in many epidemiologic studies.

5. With regard to the control of confounding factors when studying the smoking-lung cancer controversy, probably the single most common and important factor is age. And, unfortunately, the effect of age on cancer causation processes is little understood. Eminent cancer researchers believe that the aging process, in and of itself, is causally associated with the development of cancer (e.g., see Blumental, 1978).

6. There are two main types of epidemiologic studies: the follow-up study and the case-control study. Such studies have reported an association between smoking and lung cancer. However, both kinds of studies can seriously suffer from the presence of biases. The case-control study is probably the epidemiologic study design most susceptible to bias; in fact, an entire issue of the Journal of Chronic Diseases (1979, Volume 32, No. 1/2) is devoted exclusively to discussing the biases and problems associated with case-control study research. The follow-up study, in addition to the general list of biases already discussed, is subject to a special form of selection bias, namely, loss-to-follow-up; in particular, we are refer-

ring to individuals who, although initially enrolled in the study at the start of the follow-up study period, are lost during the subsequent follow-up period because of migration, death from other causes, refusal to continue participation, etc. Such losses are characteristic of all long-term follow-up studies (e.g., like those designed to study the smoking-lung cancer issue), and can seriously bias the conclusions from such studies. Greenland (1977) has illustrated the seriousness of this problem with some well-chosen numerical examples.

3. Conclusions

The belief that smoking is a cause of lung cancer can be questioned in light of the documented sources of bias attendant with epidemiologic studies of the smoking-lung cancer relationship.

Statistical associations between smoking and lung cancer, as reported in the various studies described in the Surgeon General reports, have been interpreted to mean that a causal relationship does exist. In the absence of well-designed animal and laboratory studies elucidating the meaning of these reported associations, such a quantum jump from association to causality is invalid. Indeed, the self-selection bias itself (not to mention all the other possible sources of error) is sufficient to cast doubt on the causality claim.

The main prospective (i.e., follow-up) studies concerning smoking and its relationship to mortality and morbidity from certain diseases (as discussed in the latest Surgeon General reports) do not address the issue of causality. The claim that the conclusions reached are "impressively uniform and consistent" does not mean that a causal relationship has been established, but only that approximately the same observed associations keep appearing. In fact, the biases inherent in these studies preclude the right to claim that causality has been demonstrated. Actually, one could argue that the same associations keep appearing because the same biases are present in each study.

The fact that so many studies have produced a positive association between smoking and lung cancer has led many people to make the false conclusion that "quantity means quality". The truth of the matter is that repeatability (i.e., the ability to produce the same estimated association) does not imply accuracy (i.e., the ability to produce a valid estimate of the true association). In this regard, one study free from all bias and producing a valid measure of the true smoking-lung cancer association is worth more than a thousand biased studies, all of which provide the same distorted estimate of the true association.

In summary, based on the currently available information pertinent to the smoking-lung cancer causality issue, I am of the opinion that a causal relationship between smoking and lung cancer has not been scientifically demonstrated.

Lawrence L. Kupper
Professor of Biostatistics

STATEMENT OF HIRAM THOMAS LANGSTON, M.D.
CLINICAL PROFESSOR OF SURGERY (EMERITUS)
NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

I am Hiram Thomas Langston, a thoracic surgeon in private practice in the Chicago area. I am the Chairman of the Department of Surgery at St. Joseph's Hospital in Chicago, and a Clinical Professor of Surgery (Emeritus) at Northwestern University Medical School.

I appreciate this opportunity to present my views on H.R. Bill 4957/Senate Bill 1929. I am concerned, however, with several claims made in them, especially those which endorse the hypothesis that smoking is the main cause of lung cancer.

Smoking has been said to be responsible for causing an unbelievable array of illnesses, including lung cancer. Much of the support for these accusations comes from research that is basically statistical.

Since the early 1960's, I have read most of the scientific literature on smoking and lung cancer. In my capacity as a thoracic surgeon, I do not feel qualified to respond directly to the reported statistical associations between smoking and cancer. However, I must respond to the interpretation of these associations as causal because it is inconsistent with the clinical realities of the disease that I have observed for the past forty years. Adopting the old adage "it is the exception that proves (tests) the rule," I identified certain very pertinent "exceptions" by observing firsthand the clinical behavior of lung cancer. These exceptions cast doubt upon the validity of the hypothesis that smoking causes lung cancer.

1. Inhaled cigarette smoke is equally distributed in both lungs. Why, then, as the data show, and as I have observed in my own practice, do lung cancers very rarely appear simultaneously in both lungs? The answer is not known, but this phenomenon is inconsistent with the smoking causation hypothesis.

It is of further interest to note that the vast majority of people who have been successfully treated for one malignant tumor in the lung do not develop subsequent lung tumors.

2. Cancer rarely occurs in the trachea (windpipe). The trachea is exposed to more tobacco smoke than are the lungs, because all the smoke is inhaled and exhaled through it. Also, the material deposited in the mucous lining of the air passages exits through the trachea.

The trachea is anatomically, embryologically and physiologically identical to the rest of the bronchial airway. Therefore, if cigarette smoke were a cause of lung cancer, one would also expect to see a large number of tracheal cancers. The fact is, however, that tracheal cancer continues to be an extremely rare disease.

3. Cancer of the larynx or voice box is also statistically linked with smoking. Because cigarette smoke passes through the larynx on its way to the lung, the larynx is exposed to at least the same concentration of smoke as are the lungs. Were the smoking-causation hypothesis valid, one would expect to see a rise in laryngeal cancer similar to the rise in lung cancer. Yet, the data show that there has been little change in the incidence of laryngeal cancer over the past decades.

4. I regard with a certain amount of suspicion the view that we are in the midst of a lung cancer epidemic because of cigarette smoking. Any discussion of this "epidemic" must take into account two frequently overlooked clinical factors that have had a tremendous effect on the reliability of reported lung cancer rates: (1) diagnostic techniques and (2) official certification of cause of death.

Even in the time span of my own practice, I have seen remarkable changes in our ability to diagnose lung cancer. When one considers that even diagnostic x-rays were not readily available a scant decade or two before I started practicing, it is hardly surprising that our ability to detect lung cancer has increased dramatically. And as that ability has increased, so naturally have the reported lung cancer rates.

Earlier in this century, physicians may have failed to diagnose lung cancer, resulting in rates lower than the actual incidence of the disease. Thus, when these unrealistically low rates are compared with rates for later periods when diagnostic tools were gradually becoming available, one would obtain a false impression of the real increase.

The other factor I believe to be important in evaluating whether there is a lung cancer epidemic is the accuracy of death certificate information. Death certificates are the sources for calculating death rates, but unfortunately, information in them is extremely unreliable. Most layman assume that death certificates accurately reflect the cause of death, but in many cases they do not. Coroners and non-treating physicians sign many death certificates, and they may have little or no relevant information about the actual cause of death. Even treating physicians make mistakes. That is why I have refused to consider in my own population studies any case as lung cancer unless there was microscopic confirmation of the diagnosis. Many cases lack that confirmation.

This is not to say that there has been no increase in lung cancer. I am quite convinced that a portion of it is real. I am equally convinced, however, that we have not yet identified its cause or causes.

5. In my review of the literature, I have seen the argument that the epidemiological studies show a dose-response

relationship, that is, the greater the exposure to cigarette smoke, the greater the risk of developing lung cancer. Whereas I cannot directly challenge the statistical analyses used to obtain these associations, I have been able to consider another aspect of "dose-response" -- age at diagnosis. The age at diagnosis of lung cancer does not seem to be related to the age at which a person started smoking, nor how long he smoked, nor even the number of cigarettes he smoked per day. I have observed this in my own patients, and indeed I have found it to be confirmed in the literature.

6. Age-specific lung cancer death rates almost always have a special pattern. In most series of lung cancer patients, the greatest rates occur in the 50 to 70 year age group, with a peak at 60 years. The literature also reveals that a certain generation (those born before the turn of the century) may have higher lung cancer rates than other generations.

Intrigued by these findings and the possibilities that they suggested, I reviewed approximately 4,000 lung cancer cases spanning 30 years at the Veteran's Administration Hospital in Hines, Illinois. All cases carried the diagnosis of lung cancer supported by microscopic evidence.

I found that (1) the generation born between 1890 and 1900 contributed the largest number of cases; (2) if this trend continued, this generation would fade from prominence due to old age; (3) the younger generations did not appear to be replacing this generation in cancer production. Given these points, I predicted that the number of lung cancer cases at the VA Hospital in Hines would decrease.

In a subsequent investigation of cases through 1978, I discovered that the contribution of the generation which had earlier produced the greatest number of cancers at Hines had in fact decreased significantly. In addition, the total number of cases at Hines in the period 1968 through 1978 had dropped approximately 17%. This seems to be a rather significant change which supported my earlier predictions. This observation now spans 49 years and encompasses approximately 5500 cases.

7. Lung cancer is a dynamic disease in the sense that its occurrence patterns and clinical appearance (cell type) are ever changing. For example, there appears to have been a decline in the rate of increase of lung cancer. Indeed, lung cancer incidence may have, in fact, crested. Other investigators, including some who believe that smoking causes lung cancer, seem to concur with this observation. For instance, in his address before the Health Congress in England in 1977, Sir Richard Doll said "it is encouraging to find that the total death rate from lung cancer in men decreased in 1975 albeit very slightly, for the first time in 50 years."

Perhaps what we are seeing in the case of lung cancer is what is called the "natural history" of this disease: Natural history has been succinctly described by a British thoracic surgeon as the "long drawn out process of the development and the decline of an individual disease."

If you have trouble accepting the idea that a spontaneous decline in lung cancer can occur, I remind you of the documented decline in stomach cancer. The spontaneous decline in stomach cancer over the years is a decline for which no convincing explanation has been offered. Improvements in nutrition or food storage, or diagnostic refinements, or changes in the general health of the population do not adequately explain these changes.

What explains the changes in lung cancer rates? As Dr. Belcher has pointed out, the decline in lung cancer's rate of increase started before changes in the cigarette occurred. Is this simply another example of the poorly understood natural history of a disease? Clearly, no simple explanation for these lung cancer changes appears to be forthcoming.

Many important questions about cancer causation remain unanswered. For example, precise causal mechanisms have not been identified. Many theories have been proposed, but none have won universal acceptance.

I do not agree that cigarette smoking is the major cause of lung cancer, because I believe very strongly that we do not know the cause or causes of cancer of the lung. Charges that smoking causes lung cancer are so familiar that very few people may realize that there is strong evidence to the contrary. I find that evidence to be persuasive. In my estimation, the smoking hypothesis is an oversimplification. I therefore cannot support legislation such as these two bills based upon such a questionable hypothesis.

Hiram T. Langston, M.D.

BIOGRAPHICAL DATA: HIRAM THOMAS LANGSTON, M.D.

Place of Birth:	Rio de Janeiro, Brazil
Date of Birth:	January 12, 1912 (U.S.A. Citizen by Derivative Citizenship)
Father's Name:	Alva B. Langston
Mother's Name:	Louise Foe Diuguid
Education:	Collegio Batista, Rio de Janeiro, Brazil, to 1928 Georgetown College, Kentucky 1929 University of Louisville, Kentucky, 1929-30, A.B. University of Louisville, Kentucky, 1930-34, M.D. University of Michigan, Graduate School, 1939-41, M.S. (Surgery)
Married:	Helen M. Orth
Date of Marriage:	June 22, 1941
Children:	Paula F. Langston, born June 15, 1946 Thomas O. Langston, born September 5, 1949 Carol E. Langston, born September 4, 1953

BRIEF CAREER SUMMARY

Born of U.S. parents engaged in educational missions under the Southern Baptist Convention. Educated through sophomore year of college in Rio de Janeiro, subsequent education as outlined above. Elected to Theta Kappa Psi medical fraternity and Alpha Omega Alpha honorary medical fraternity. Internship: Garfield Memorial Hospital, Washington, D.C., 1934-35; Resident in Pathology, Garfield Memorial Hospital, 1935-37; Assistant Resident in Surgery, University Hospital, Ann Arbor, Michigan, 1937-38; Resident in Surgery, University Hospital, Ann Arbor, Michigan, 1938-40; Instructor in Thoracic Surgery, University Hospital, Ann Arbor, Michigan, 1940-41; Private Practice and Associate in Surgery; Northwestern University, Chicago, Illinois, 1941-42; February 1942 to February, 1946, Military Service, Northwestern University sponsored hospital (12-th General). Served in North Africa and Italy as Chief of Thoracic Surgery. Rose to rank of major, AUS; ultimately awarded Army Commendation Ribbon, Bronze Star Medal and Ordem do Merito Aeronautico (Officer Grade) (Brazilian Air Force). Returned to Northwestern University with rank of Assistant Professor of Surgery. In 1948 entered private practice of Thoracic Surgery in Detroit, Michigan, and served as Associate Professor of Surgery at Wayne University. Returned to Chicago in 1952 with Appointments as follows: 1. Associate Professor of Surgery at University of Illinois College of Medicine. 2. Chief Surgeon, Chicago State Tuberculosis Sanitarium, Department of Public Health, State of Illinois, 1952-71. 3. Consultant in Thoracic Surgery to the Veterans Administration Hospital,Hines, Illinois. (1974) On the staff of Augustana (to 1971), Gottlieb, Grant and Saint Joseph Hospitals.

Appointed Clinical Professor of Surgery at the University of Illinois, College of Medicine - 1962 and Professor of Surgery, Abraham Lincoln School of Medicine, University of Illinois, 1973 to 1977 - Served as Chief of Thoracic Surgery (General) Abraham Lincoln School of Medicine, University of Illinois, 1973 - 1975. Attending Thoracic Surgeon - Cook County Hospital 1966 - 1977. Appointed Clinical Professor of Surgery, Northwestern University Medical School - 1978-1981, Emeritus 1981

PRINCIPAL PROFESSIONAL OR BUSINESS ACTIVITIES

Diplomate, American Board of Surgery, 1942 - Founder Member, Board of Thoracic Surgery, 1948; Member, the Board of Thoracic Surgery, 1956-1961; Member of various committees of national and local societies - Secretary, American Association for Thoracic Surgery, 1956-1961; Vice President, American Association for Thoracic Surgery, 1968-1969; President, American Association for Thoracic Surgery, 1969-1970; Vice-President, Chicago Surgical Society, 1971-1972; President, John Alexander Society, 1977. Chairman, Department of Surgery, Saint Joseph Hospital, 1977 to Present.

Member, Editorial Board of the John Alexander Series, Charles C. Thomas, Publisher, 1957-1978; Member, Editorial Board, Journal of Thoracic and Cardiovascular Surgery, 1962-1978; Member, Residency Review Committee - Thoracic Surgery - Representing American College of Surgeons, 1967-1972; Chairman, 1969-1972. Vice President Staff, Grant Hospital, 1972 to 1975; Member, Board of Directors, Grant Hospital, 1973-1975. Thoracic Surgery Representative, Interspecialty Council AMA, 1972-1976; National Thoracic Surgery, Manpower Study - Executive Committee, 1971-1973. University of Illinois Distinguished Service Award, June, 1975. (Department of Surgery). Chairman, Membership Committee - Chicago Surgical Society, 1979. Member, Standards and Ethics Committee, Society of Thoracic Surgeons, 1979-82. Delegate (Alternate) House Delegates, AMA - representing American College of Surgeons, 1978-1980. Chairman, Long Range Planning Committee, Saint Joseph Hospital, 1978 to 1980. Member, Subcommittee on Medical Practice, Chicago Medical Society, 1980.

Membership in Medical Societies

American College of Surgeons
American Association for Thoracic Surgery
American College of Chest Physicians (Fellow)
American Medical Association
American Surgical Association
Chicago Medical Society
Chicago Surgical Society
Illinois State Medical Society
Illinois Surgical Society
The Institute of Medicine of Chicago
Pan-Pacific Surgical Association
Society of Thoracic Surgeons
Western Surgical Association
Societe Internationale de Chirurgie
American Thoracic Society
Illinois Thoracic Society
Samson Thoracic Surgical Society (Honorary) 1980
Illinois Thoracic Surgical Society (Honorary) 1981

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Biographical Data of Hiram Thomas Langston, M.D. - 3

Membership on Hospital Staffs

Clinical Professor of Surgery, Northwestern University
Medical School, 1978-1981, Emeritus 1981
Clinical Professor of Surgery, University of Illinois
College of Medicine - 1952-1978
Chief of Surgery, Chicago State Tuberculosis
Sanitarium, Department of Public Health,
1952-1971, State of Illinois
Consultant in Thoracic Surgery, Veterans
Administration Hospital, Hines, Illinois - 1952
Attending Physician in Thoracic Surgery,
Cook County Hospital - 1970-1977
Grant Hospital
Henrotin Hospital - Resigned 1973
Augustana Hospital - Resigned 1971
Lake Forest Hospital - Consultant
Saint Joseph Hospital
Highland Park Hospital - Resigned 1971
Visiting Professor, Rush Medical College -
Consultant Presbyterian St. Luke's Hospital
Lecturer - Abraham Lincoln College of Medicine -
University of Illinois -1978

Visiting Professorships

Mayo Clinic, Rochester - Sept. 27-29, 1966
University of Missouri - Columbia - Feb. 8-9, 1971
University of Florida - Gainesville - Sept. 22-24, 1971
University of Michigan - Ann Arbor - May 16-18, 1973
Chicago Medical School, April 9, 1980

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   with William M. Tuttle, M.D. and Robert T. Crowley, M.D.; Abstract in the Medical Bulletin of the Mediterranean Theater of Operations, 2: 142-149, December, 1944.

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The Alleged Costs of Cigarette Smoking

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House of Representatives Bill No. HR4957, introduced by Mr. Waxman on November 12th 1981 contains on page 2 the following passage :

> "(8) it is estimated that cigarette smoking related deaths and disabilities result in $25,800,000,000 annually in lost productivity to the United States economy and $13,600,000,000 in medical costs."

As an applied economist with experience in cost-benefit analysis, I have been interested in this and related issues for several years. As I indicate below, there are numerous problems in making statements of the kind just quoted. In my experience, most of these statements are based upon inadequate economic analysis and unreliable statistical data. As a result, they tend to be seriously misleading, therefore it seems worthwhile to set out briefly some of these difficulties involved.

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1. It is by no means established that cigarette smoking is a cause of illness and death as claimed in items (1) to (6) of HR4957. Since this is not my area of expertise I shall not dwell on it further. But the point to establish is that the reliability of any cost calculations cannot be any greater than the reliability of the underlying medical diagnoses. If the medical experts cannot agree on the effects of smoking, either in general or in specific instances, it follows that there cannot be agreement on the costs of smoking. Indeed, if one takes the view that smoking is statistically associated with certain illnesses but is not the cause of those illnesses, then the value of lost productivity and medical costs properly attributable to cigarette smoking is zero.

2. Even if cigarette smoking is claimed to be a cause of illness and premature death, the extent of any such contribution remains uncertain. There are inevitably a number of contributory factors associated with the incurring, severity and duration of any illness and with the timing of death. For example, personal habits of diet and exercise, as well as personality factors, influence the onset and severity of certain illnesses, which in turn affect the number of days off work or spent under medical care. Even if smoking were

a contributing factor to illness and premature death, it would be quite inaccurate to attribute to smoking the whole of the associated costs. But exactly what proportion ought to be attributed is unclear, partly because the extent of any contribution of smoking is uncertain, and partly because there is no basis in pure logic for attributing joint effects among the various possibly contributory factors.

To illustrate, specific examples of this point are found in items (5) and (6) of HR4957, which claim that "women who take birth control pills and smoke are more likely to suffer a heart attack or stroke" and that "certain occupational hazards in conjunction with an individual's smoking increase substantially the risk of disease and death". The first difficulty is an empirical/medical one : how far is it possible to identify and measure accurately the separate effects, if any, of pills and smoking, or occupational hazards and smoking ? The second difficulty is one of principle : how far should any combined effects of pills and smoking be attributed to smoking rather than to taking birth control pills ? If, to take a hypothetical case for illustrative purposes, it were established that taking a specific pill and smoking had no effect separately, but had a specific effect when combined, it would be quite arbitrary to attribute the effect (and its consequent costs) to smoking alone.

3. People engage in an enormous variety of activities some of which may result in accidents, illness or death, thereby reducing productivity and generating medical costs. Dramatic recreational examples include skiing, mountain-climbing, potholing (cave exploring), and skydiving; more mundane examples include travelling by car or plane, or over-indulgence in food, drink, physical exercise, sunbathing, nightlife and so on. All these activities generate costs in some cases presumably of substantial magnitude, but while these costs are to be regretted, it is recognised that they do not constitute the whole of the story. There are corresponding benefits to be taken into account, which in the eyes of those who engage in these activities more than compensate for the costs incurred. Calculations of the purported costs of cigarette smoking generally take no account of the benefits perceived by smokers. In this respect, cigarette smoking is being singled out and treated differently, for reasons that are not made clear.

4. Calculations of the claimed costs of smoking frequently assume that if people were not affected by illnesses allegedly caused by smoking they would otherwise be perfectly healthy.

This, of course, is not the case : They are typically affected by a variety of illnesses. The relevant loss in productivity is thus related not to the total absence from work experienced by smokers, but to the incremental absence, if any, that can properly be attributed to smoking. This would be naturally much lower.

5. Similarly, studies often calculate the total medical costs incurred by smokers. This is incorrect : the appropriate calculation is the incremental cost, if any, attributable to smoking. In particular, it needs to be taken into account that nonsmokers who live longer tend to generate very substantial medical costs in old age. Indeed, several studies by economists sympathetic to the anti-smoking cause have concluded that the net medical cost of smoking is negative - that is, the lifetime costs of treating smokers are in fact less than the lifetime costs of treating non-smokers.

6. Finally, it might be thought that the costs of smoking are incurred not by the smoker but by the rest of society, so that smokers are effectively subsidised by non-smokers. This is far from the case. Any lost productivity due to absenteeism or ill-health is probably reflected in lower wages, and medical costs are frequently born by the patient (possibly in the form of higher insurance premiums). But any such costs are far

outweighed by the heavy taxes on tobacco products. There is no doubt that, on balance, there is a transfer of income from smokers to the rest of society.

For these six main reasons, calculations of the claimed costs of smoking are fraught with difficulties, and those that I have studied have generally proved inadequate and misleading. It is possible that the figures embodied in HR4957 are based upon new and more acceptable calculations, but in the absence of such evidence I am inclined to view them with suspicion.

Statement of Eleanor J. Macdonald
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My name is Eleanor Macdonald, Professor Emeritus of Epidemiology at the University of Texas System Cancer Center, M.D. Anderson Hospital and Tumor Institute, Houston. I have worked in the field of epidemiology for over 40 years in three state programs in Massachusetts, Connecticut and Texas; in fact, I established the first state cancer registry in the U. S. From 1948 through 1974, I chaired the Department of Epidemiology at the University of Texas System Cancer Center. I have authored or coauthored about 150 publications, most of which deal with different phases of cancer epidemiology, and I remain actively involved in a number of ongoing research studies in epidemiology. I am also the editor of the Epidemiology, Statistics and Cancer Control section of the *Yearbook of Cancer*.

I share the concerns of this Committee regarding cancer morbidity and mortality, and I encourage legitimate efforts to control this disease. However, as a scientist who has dedicated her professional career to a careful study of cancer epidemiology, I am appalled at the belief implicit in H.R. 4957, H.R. 5653, and S. 1929 that Congress can legislate scientific fact. I urge this august body of well-intentioned legislators to avoid putting itself in such an untenable position.

These bills are a misdirection of governmental energy and purpose because of their narrow focus on smoking as the major cause of lung cancer. We do not, in fact, know the cause or causes of lung cancer and it is not in the best interest of either science or government to take a simplistic approach to the task of identifying them. There are obviously many factors in cancer causation that we have not yet begun to understand. For example, the depth of our uncertainty is still far too great regarding the effects of nutrition, work history, life style, ancestry and environment.

One of the main reasons that I disagree with these bills' findings regarding smoking and cancer stems from my knowledge of the primary type of evidence on which these judgments are based, i.e., epidemiological. As an epidemiologist who has conducted large-scale population studies, and who has reviewed the data of vast numbers of research reports, I am perhaps more aware than many scientists -- and certainly more aware than most laymen -- of the limitations of this type of data. Congress must understand that epidemiology is not an exact science, and no amount of wishing can make it so. We cannot turn humans into animals directed at will toward various exposures.

We cannot send all death certificates to one infallible pathologist for correction and confirmation. We cannot force all individuals supplying the raw data to epidemiologists, or even all epidemiologists themselves, to demand the type of precision necessary in data gathering and review to insure that the quality of the data meets high scientific standards. Consequently, judgments about chronic disease causation based on epidemiological findings must be highly tentative; they do not enjoy the absolute certainty implied in the findings of these three bills.

Mortality data are frequently used by epidemiologists to study disease trends, often without full awareness of inherent weaknesses in these data. Mortality rates are obtained from information on death certificates furnished by physicians or local health officials. Although I am certain that every effort is generally made to fill these out conscientiously, errors do occur -- in diagnosis as well as in recording of the data.

In addition, other exogenous influences can cause profound fluctuations in mortality data over the years that can easily be misinterpreted as changing disease trends. For example, the introductions of each of the seven revisions of the International Lists of Diseases and Causes of Death (ICD) have had effects, generally improvements, on the classification of diseases. With each revision, however, the continuity of the disease trends has been broken and in some cases seriously distorted. Sadly enough, even though it has taken us centuries to attain the classification system that we now have, it is still imperfect.

The possibility of shifting classifications bringing about a rise or fall in certain causes of death makes some investigators question the value of any effort to compile statistics on causes of death. For example, anyone attempting to trace the patterns of lung cancer during the twentieth century must remember that the 1948 ICD revision, first applied in 1949, introduced a classification separating primary cancer of the lung from metastatic cancer of the lung (coded thereafter as "unspecified as to primary or secondary). For two years, there was a sharp reduction in the reported primary lung cancer rate, and then a rise in both the primary and unspecified rates. How long did it take for the new classification to catch on? How accurately did physicians distinguish between primary and secondary? When epidemiologists study twentieth century lung cancer trends, do they include or exclude the unspecified and secondary cancers? We cannot be sure about the answers to these questions.

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The 1965 ICD revision, adopted in 1968, seriously complicated the situation by deleting the unspecified category. This revision effectively combined primary and unspecified lung cancer, thereby removing a needed safeguard for accuracy. Primary and secondary lung cancer are separate disease entities, with quite possibly distinctly different causes. Since ten percent of all cancers spread to the lung, and since, for nearly twenty years, more deaths were coded in the unspecified than in the primary category, the combination of those two categories seriously confused the lung cancer data. The damage done to epidemiological investigations of lung cancer by the 1965 classification change cannot be underestimated. In the ninth revision (1980), greater confusion was added by placing the bronchus and lung unspecified as the ninth subdivision of primary lung cancer (162.9). Death certificates seldom list more than a three-digit number, such as 162. We simply cannot separate out from the mortality data the specific information we need to study lung cancer scientifically.

Another aspect of the problem of changing disease classifications can be found by comparing trends in stomach and lung cancer. In the first half of this century, the phrase "question of gastric cancer" was used frequently on death certificates when a cachectic patient died within days or hours of first summoning a physician. It is fascinating to see that in

47 states, within two years of the introduction of the 1948 revision, the reported gastric cancer mortality rates dropped as suddenly as the unspecified lung cancer ones rose. Thus, the two changes may in part represent a shift of questionable diagnosis from the difficult to diagnose stomach cancer to the equally difficult to diagnose lung cancer classification.

Besides studying anomalies that have developed in overall lung cancer trends, I have also examined trends in the reported lung cancer mortality rates for women in this century. For years, those individuals who believed that smoking caused lung cancer focused almost solely on male lung cancer, and generally tended to ignore female lung cancer. In recent years, however, more attention has been given to female lung cancer because of reports of an increase in female lung cancer and the claim that increased cigarette smoking was responsible. I do not accept this interpretation because I have serious scientific reservations about both the accuracy of the underlying data and the conclusions drawn from it.

First of all, we simply do not have reliable data on smoking prevalence in women -- or in men, for that matter. The best information we have is from the National Health Survey reports, yet the 1978 report, for example, has a standard error of 30 percent. That percentage of error is too high to provide a basis for drawing definitive conclusions.

Secondly, I have doubts about whether lung cancer in women has actually increased dramatically. As I indicated earlier, national mortality data may not be reliable. If we assume for the sake of argument, however, that they are reliable, we can still question the argument that female lung cancer has increased sharply in recent years. Although the age-adjusted U.S. death rates from respiratory cancer in white females show a consistent increase from 1953 through 1975, the rate of this increase has varied over this period. A smoothed year-to-year variation in the rate of increase -- the slope -- indicates that a sharp rise began around 1960, but leveled off around 1970. Although the rate of increase has begun to rise again, it is not as steep an ascent as in the 1960's.

This is important because the slope gives a predictive picture of the overall mortality trend. And the slope that I see for female lung cancer suggests a stabilization. It may even predict a decline in lung cancer mortality in the coming years.

Other factors must also be considered in an analysis of the apparent increase in female lung cancer. One such factor, inaccurate reporting, appears to have played a predominant role.

My analysis of both reporting techniques and the underlying data suggests that lung cancer in women was underreported in the past and is being overreported now.

Underreporting may have occurred primarily because of three clinical factors: 1) less clinical interest in female lung cancer, 2) inadequate diagnostic tools, and 3) clinical misdiagnosis of lung cancer as tuberculosis or other respiratory diseases. First of all, for many years, clinicians tended to believe that women were less likely than men to develop lung cancer, and that belief could have affected their diagnoses. Second, physicians were less capable of detecting lung cancer, because they simply did not have adequate means of discovering the presence of a cancer. Third, mainly as a consequence of these limitations, physicians may have confused lung cancer with other respiratory diseases, particularly tuberculosis. In other words, they may have diagnosed tuberculosis when in fact the patient had lung cancer.

The introduction of antibiotics and other drugs led to a sharp decline in deaths from tuberculosis and other infectious diseases. With isoniazide and these other drugs at their disposal, physicians could suddenly separate the lung cancer patients from the tuberculosis patients, because the TB patients generally improved after drug therapy whereas the cancer patients did not. The abrupt drop in tuberculosis mortality rates after the introduction of isoniazide in 1952, and the nearly simultaneous increase in reported lung cancer rates, provide substantial support for my belief that these diseases were often confused clinically. Therefore, as these drugs became increasingly available, underreporting of female lung cancer became less common.

Unfortunately, there is evidence which suggests that today lung cancer in women is overreported. This may occur in one of two ways: (1) clinicians are not always able to distinguish between primary and secondary lung cancer. As discussed earlier, the distinction is of obvious importance for a factual count of primary lung cancer cases. The only truly effective way to determine whether lung cancer is primary or secondary is through autopsy, and few cases of cancer deaths in this country are autopsied. In general, the autopsy rate is about 12 percent. When one considers that this percentage includes all the accidental and violent deaths that must be autopsied, one understands just how low the autopsy rate for lung cancer may be. This issue becomes even more crucial because primary lung cancer is one of the more difficult diagnoses to establish clinically. (2) The current ICD classification combines primary and unspecified lung cancer into one category. Even if the clinician could correctly distinguish primary from unspecified lung cancer, the effort would be negated by classification methods. Thus, both primary and unspecified lung cancers are combined in the reports that have described increases in lung cancer in women.

Therefore, changes in diagnostic techniques and changes in disease nomenclature are very important because, speaking from the epidemiologists' point of view, we cannot be sure that the reported trends in lung cancer among women accurately reflect the true incidence of the disease.

Further support for my skepticism regarding an identification of cigarette smoking as the primary cause of lung cancer stems from my own research investigations of this disease. In one, a study of lung cancer in Spanish surnamed and Anglo women in El Paso, we found a high incidence of the disease in the Spanish surnamed, 64 percent of whom were smokers. Although the same percentage of the Anglo women also smoked, their rate of lung cancer was half that of the Spanish surnamed. We concluded that we had to rule out smoking as the significant factor. Instead, the most important factor in the Spanish surnamed women's lung cancer was their residence from birth to adulthood in adobe houses. These are solidly built, nearly airtight and poorly ventilated structures heated mostly by wood fires, which exposed the women to known carcinogens.

In a much larger study, my colleagues and I examined the relationship of environmental and ethnic factors to cancer mortality by site for every state and region of the U. S. for a twenty year period. We began with a very large number of variables or factors, from which we selected 12, among them cigarette consumption, intake of different kinds of alcoholic beverages, ethnic background, pollution, income, temperature, etc. We used these to derive sophisticated statistical models of the geographic patterns of cancer mortality in the U. S. These models were an attempt to describe by statistical equations the relationships between certain factors and cancer mortality.

We found that all the factors used in the study accounted for only 36 percent of the geographic variation in white female death rates from cancer of the bronchus and lung specified as primary. The best model -- which eliminated the unimportant factors and brought into focus the important ones -- accounted for 29 percent. (In contrast, the models accounted for 89.5 percent of the variation in cancer of the breast.) This indicates that unknown but crucial factors were not included in the variable pool from which the model for primary lung cancer was derived. Cigarette consumption, which was included, turned out not to be a factor.

We also discovered a warping in the basic data dividing primary lung cancer from the unspecified category of lung cancer -- for both white females and white males. This is important because it shows we can't be sure lung cancer rates, as reported, present a factual picture.

There are other important considerations required in a balanced discussion of lung cancer in women. For instance, women in increasing numbers have, over the last 35 years, left the jobs they have traditionally performed and have entered work areas formerly reserved for men. Women are now exposed to the same community and work environments as men, and there are many unknown carcinogenic potentials in these environments.

The tremendous variety of toxic and carcinogenic airborne chemicals produced by industry and released into indoor and outdoor environments, in our homes and our workplaces, suggest a set of factors which may be important in lung cancer etiology. In 1976, I published a 30 year study which examined the possible influence of the petro-chemical industry exposures in Houston upon lung cancer mortality, heart diseases, and other respiratory diseases. In certain areas clustered around industrial facilities, mortality rates from respiratory cancer and other diseases were found to be very high -- so high, in fact, that deaths from respiratory diseases nearly doubled over the last 15 year period. Outside the industrial "pathway," mortality rates had remained stable for twenty years. A similar finding was made by Clemmesen in an industrial city in Denmark. These epidemiological findings are strongly suggestive of the fact that industrial exposure factors underlie the etiology of lung cancer. I might add that I saw no evidence that cigarette smoking could explain the peculiar disease patterns I found in Houston.

Our increasing ability to identify industrial carcinogens has opened many avenues of study and emphasized the importance of industrial environments. Control of these carcinogens at their source offers the most positive potential for prevention of lung cancer.

To date, epidemiological studies of lung cancer have not generally included adequate information about occupational exposures. Frequently, an epidemiologist may get little more than a brief indication of occupation -- such as, "construction worker." This tells us nothing about that person's actual exposure. At the M. D. Anderson, one of the major centers in the U. S. for the study and treatment of cancer, records are even less informative: the vast majority of cancer patients' records simply list "retired" as the occupation. How helpful, then, can we expect the records of private practitioners and community hospitals to be? Even when attempts are made to elicit full occupational exposure history, the patient's knowledge and/or memory of previous exposure will undoubtedly be faulty and incomplete. I seriously doubt if the typical industrial worker has an exact record of the hazardous substances, much less the amounts, to which he may have been exposed over a 30-40 year time period. And his wife, who may well describe her occupation as housewife, would undoubtedly know even less about the substances she cleaned from his work clothes all those years.

Still other important areas of research, besides the above mentioned, are emerging in our study of the puzzle of cancer causation. Scientists are beginning to recognize the possible roles played by nutritional imbalances in the etiology of cancer. Although this discipline is still in its infancy and conclusions do not appear to be immediately forthcoming, this new area of research does point to the fact that there exist many scientific unknowns in the study of cancer causation.

From my own studies and from a considerable amount of other research reported in the literature, it is therefore apparent that calling cigarette smoking the single major causative factor in female lung cancer is an oversimplification of a very complex epidemiological problem.

Conclusion

There is a concept widely accepted by public health schools that if many studies, in themselves not based on definable populations, all arrive at the same conclusion, then that conclusion must be valid. This concept disregards the fact that the same intrinsic error in method might produce the same result.

In the first and subsequent Surgeon General's reports, one of the main sets of supportive data was based on the survey of the American Cancer Society. The inadequacy of this survey was recognized and critically analyzed by many of the foremost statisticians and epidemiologists at the time, including Sir Ronald Fisher, the father of statistical methods as we now practice them, Dr. Joe Berkson of the Mayo Clinic, who devised the method of reporting survivals we all use, Professor K. A. Brownlee, for the American Statistical Association, Dr. Donald Mainland, physician and teacher of medical statistics, and numbers of others.

In the Cancer Society study, thousands of women volunteers asked of their acquaintances many questions about their personal habits. No one questions the sincerity of the women, but no business would consider basing its sales policy on the results of such a study. The proportion of smokers interviewed bore no resemblance to the proportion of smokers in the population, which demonstrated that the women surveyors went out to find smokers. Twenty-five states were not in the study. It was largely an urban New York, New Jersey population, of upper middle class people.

An enormous literature has grown up on the subject of smoking. But the bulk of the evidence is statistical. In no single instance has cancer of the lung been produced in any meaningful animal experiment using tobacco smoke inhalation, even though millions of dollars have been spent in the effort.

In all my years as a cancer epidemiologist, I have seen many theories studied and debated. But only in the instance of the smoking theory has the theory itself become sacrosanct. Any of the many valid scientific observations which refute the theory are discounted as bizarre. Many a doctorate on human psychology will be earned in the future, when the actual cause of lung cancer becomes known, which will attempt to explain the intensity of the promotion of this unproved theory.

Science, by explaining one fragment after another of genetic, biochemical, somatic, behavioral, environmental, and nutritional information, is building gradually the structure, which, upon completion, will help us understand the genesis of cancer. It is becoming increasingly clear that there is no single, simplistic answer to the question of what causes respiratory cancer. It is hardly in the best interest of either science or government to create the illusion that an attack on a single lifestyle factor will provide the solution for such a complex problem. The proposed legislation creates this illusion and should not be passed into law.

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STATEMENT OF JOHN E. O'TOOLE, CHAIRMAN, FOOTE, CONE & BELDING COMMUNICATIONS, INC.

Mr. Chairman, members of the subcommittee. Thank you for allowing me to appear today in this consideration of H.R. 5653.

My name is John E. O'Toole. I am chairman of the board of Foote, Cone & Belding Communications, Inc., the fourth largest U.S. advertising agency -- ninth largest worldwide -- with 34 offices in 19 countries. Our New York office works on special product assignments for Lorillard and one of our Chicago accounts is Brown & Williamson International Tobacco. Foote, Cone & Belding is the successor agency to Lord & Thomas, one of the oldest U.S. advertising agencies, which for many, many years from the turn of the century worked with the American Tobacco Company.

Before I address the proposed legislation, it would seem appropriate to discuss for a moment the theory and purpose of advertising. Eighty years ago the generally accepted definition, was "keeping your name before the public." In 1904, a bright young employee of Lord & Thomas, Albert Lasker, and a former Royal Canadian Mounted Policeman named John F. Kennedy who became a Lord & Thomas copywriter, redefined the mission of advertising in a way that was to affect our industry and indeed all of those industries offering the public goods and services from that day to this.

Advertising, as Lasker and Kennedy defined it, is "salesmanship in print." This realization changed the course of advertising completely. It added the concept of persuasion, the prime role of the salesman, thus producing a creative explosion that made the advertising industry a potent force in the American economy and taught businessmen throughout the world how advertising could move their products and stimulate the economies of their own cities and nations.

Advertising, then, is salesmanship, functioning in the paid space and time of mass media. It is based on the information with which a salesman must be equipped to make a person-to-person sale. In the old days it might have persuaded a consumer to buy a new type of product, as Albert Lasker did with American Tobacco's new blended cigarette. Today, however, cigarette advertising persuades the smoker to try another brand. It does so on the basis of what our research tells us smokers are seeking in a cigarette -- low tar, perhaps, or flavor, or the right balance of each.

I[illegible] a smoker, try a pack of cigarettes not usually my brand, because a magazine ad has persuaded me that it is richer and fuller in flavor than those I now smoke, I do not have to buy a second, third or fourth pack if I find the product not up to my old one. Through advertising I make an informed choice to try a product.

It has been said that the new rotating labels proposed by H.R. 5653 will enable the smoker to make a more informed choice. And here we come to the crux of my testimony here today. Say that it is decided that a brand of cigarettes selected to carry what I will call the pregnancy warning is my brand. Say that among the brands that must under the system to be created by the Federal Trade Commission carry for the first year what I shall call the heart disease warning is the favorite of my next-door neighbor and friend. Now I am not of a mind to bear a child. Much as my wife and I love our two daughters, we do not really care to add another child to our family now. And my neighbor, who has the lowest blood pressure on the block, knows of no one in his family for generations back with any hint of heart disease. If I read on my pack everty time I reach for a cigarette that smoking and pregnancy don't mix -- if my neighbor makes mental note that the Congress has mandated the heart warning every time he picks up his pack -- are we being informed of anything that is pertinent to us as smokers? Aren't we and millions of smokers to whom the warnings on their favorite brands have little or no relevance in effect being misled? If, conversely, I am worried about heart disease because I know of a family tendency toward coronary occlusion, need I shop for the brand whose advertising does not mention diseases of the heart? Should the woman, who is especially conscious of cancer because her mother, or best friend, or a person with whom she works have all had cancer, search for a brand that speaks only of heart disease?

When the Congress decided in 1965 that cigarettes should be labelled as potentially dangerous -- and when the warning label then appeared in advertising in 1972 -- it was, as the 1965 legislation states, that "the public may be adequately informed that cigarette smoking may be hazardous to health." From all accounts, except the FTC's, the label on packs and in advertising has been effective in spreading that word. Would it not be, in effect, deceptive advertising now implicitly to tell the smoker that he is in danger of heart disease or she chances a spontaneous abortion by smoking specific brands of cigarettes? This is a gamble that I do not think the Congress, or indeed the FTC, which has for several years advocated rotating explicit warnings, should take.

As an advertising man of 30 years' experience, I am worried, too, about another gamble involved in this bill. Very few advertising copy themes, illustrations or headlines nowadays get into public print or on the air without adequate research, without the principals being convinced that the message and all its connotations will be received in the manner in which the advertiser intended it. The testimony and questioning of witnesses here a week ago has revealed that there has been no research on the effect of the various warnings proposed in H.R. 5653.

Testimony before the full Commerce Committee in 1965 from psychology and marketing experts on the originally proposed warning label warned of the dangers of overstatement. One witness, the chairman of the department of marketing at Notre Dame, told the committee that to overstate the dangers of smoking could lead to an undermining of the effectiveness of warnings in use in connection with other products. "We all know the story of the boy who cried wolf," he said. "If warnings on cigarette packages portrayed smoking as a more serious or immediate danger than our experience confirms, we would soon come to attach little significance to the warnings."

A licensed psychologist and associate dean of the graduate school of business administration at Michigan State University went even further in his caution to the committee. Referring to the FTC's proposed warning requirement then in 1965, with its reference to "death from cancer and other diseases," he said, "In my judgment, a warning expressed in such language would, by virtue of its distastefulness and intended shock effect, precipitate a far greater backlash than a quieter, more restrained warning statement."

His fear was echoed in 1977 by psychologist Daniel Horn, the longtime head of the government's anti-smoking bureau, the National Clearinghouse for Smoking and Health, and a pioneer in education against smoking. Dr. Horn said, "I was always for the health warning [but] I was always concerned really about the level of strength in the health warning, partly because if a warning is too strong it becomes counterproductive." He added that he did not see the health warning as "an important source of education."

I should like to conclude with some comments from a book I wrote last year called "The Trouble With Advertising...," a book in which I presented an affirmation of advertising arrived at by means of a critical view from the inside. The portion I would like to tell you about is from a chapter entitled "What Advertising Isn't," in which I noted a 1979 FTC staff report, "Consumer Information Remedies." What Dr. Horn would call "education" is what the commission staff writers call "consumer information." These staffers, in discussing how to evaluate consumer information, wrote:

"The Task Force members struggled long and hard to come up with a universally satisfactory definition of the value of consumer information. Should the commission consider a mandatory disclosure to be a valuable piece of information, for instance, if it were later shown that although the consumers understood the information, they did not use it when making purchasing decisions? Is there a value in improving the quality of market decisions through the provision of relevant information, or is it necessary for the information to change behavior to have value?"

I wrote that "the ensuing 'remedies' make it clear that the staff really judges the value of mandatory disclaimer by the degree to which it changes consumer behavior in the direction they are seeking."

I pointed out that I'm a consumer and I resent government officials wondering what to do with me next if I understand but choose to ignore a disclaimer they've forced an advertiser to put in his ad? It's my God-given right to ignore any information any salesman presents me with -- and an ad, remember is a salesman. What's this about changing behavior? "Well, mine," I wrote, "is going to change if the employees of a government I'm paying for are talking like that out loud. It's going to get violent," I wrote.

These FTC persons -- indeed, with all due respect, Mr. Chairman, perhaps even you and the members of the subcommittee -- do not understand the nature of advertising, just as the professional critics of advertising, the journalists, consumerists, academicians, don't understand that advertising is not journalism or education and cannot be judged on the basis of objectivity and exhaustive, in-depth treatment.

Thorough knowledge of a subject cannot be derived from an advertisement but only from a synthesis of all relevant sources: the opinions of others, reports in newspapers, magazines and, increasingly, television -- even the advertising of competitors, which in the case of cigarette advertising gives the smoker the information he may seek in terms of tar and nicotine levels. I might add, too, that it is not just the warning on the pack and in advertising that has gotten across the message that the Congress decided upon, first in 1965 and then in 1969. It is the efforts of the American Cancer Society, the American Lung Association and the American Heart Association in spreading abroad the purported dangers of smoking.

Mr. Chairman, members of the subcommittee, I thank you for your time and patience. I hope you will remember when it comes time to vote upon H.R. 5653 that thorough knowledge of a subject cannot be derived from an advertisement and that by all reports, the American public is already thoroughly aware that smoking may be dangerous to health. Listing specific dangers of individual brands can only lead to overkill and discriminate against manufacturers of those brands which, in the roulette system to be devised by the Federal Trade Commission, may be forced to bear the label most explicit -- and most relevant to most smokers.

Statement by
L. G. S. Rao, Ph.D.
Bellshill Maternity Hospital
Bellshill, Scotland, U.K.
Regarding H.R. 4957
S. 1929

My name is Dr. L.G.S. Rao; I am Senior Biochemist at Bellshill Maternity Hospital in Glasgow, Scotland. I obtained my Ph.D. in Biochemistry from the University of Newcastle in 1966. I am the author of numerous scientific publications and have made presentations at scientific meetings in Europe and the United States.

My experience in clinical biochemistry over the past 20 years has been varied and has resulted in the development of interests in several methodological and clinical problems including perinatal medicine. Over the past several years, I have become deeply interested in the investigation of the causes of the high incidence of low birth weight and perinatal mortality which is found among the poorer patients of Bellshill Maternity Hospital. I have attempted to define in biochemical terms the risk factors associated with the "poor social conditions" which are supposed to be the cause of the poor reproductive performance of these mothers. I have found, as have other researchers in this field, that the biological characteristic of the poorer social groups that is of relevance to fetal growth retardation is their poor nutritional status and not, as has been claimed, their smoking habits. This finding could be of considerable practical importance because nutritional deficiencies can be corrected by dietary advice or supplementation. A program of such dietary intervention could lead to a striking reduction in the incidence of low birth weight and perinatal mortality and morbidity. Set forth below is a review of the scientific research supporting the above conclusion:

Introduction

There is a widely held view that maternal smoking is a cause of low birthweight (LBW) and perinatal mortality* (PNM) (the "causal" hypothesis). That view is challenged herein because the statistical association between smoking and LBW and PNM lacks the specificity and the consistency to be of causal significance and because of evidence inconsistent with the causal hypothesis.

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*The term perinatal mortality includes both stillbirths and infant death within the first few weeks after birth.

Most of the studies which favour the causal hypothesis have not corrected for factors which are already known to have a marked effect on LBW and PNM. The most conspicuous fact that emerges from a scrutiny of all the studies on smoking in pregnancy is that the so-called "effect" of smoking is seen only in the poorer underprivileged mothers and not in the mothers who have a good family income. Low family income could lead to nutritional deficiencies which can cause fetal growth retardation. Therefore, the so-called "effect" of smoking seen in only the poorer mothers may not be due to smoking itself, but due to deficiencies in maternal nutrition during pregnancy. Evidence for this has come from a recent study on the protein intake in pregnancy in Bellshill Maternity Hospital which showed that among mothers with normal protein intake, there was no difference in the proportion of LBW infants between smokers and non-smokers.

Other evidence against the causal hypothesis from a review of the available literature is also presented.

Smoking in pregnancy has become such an emotional issue that it is difficult to be unbiased in the design of the studies on this subject or in the interpretation of the results. As there is a great deal of evidence against the "causal" hypothesis, other hypotheses are required to explain the higher incidence of low birthweight infants and higher perinatal mortality found in some studies on smoking and pregnancy. There is considerable evidence for the view that fetal growth-retardation is mediated through a reduced weight gain in pregnancy, possibly due to undernutrition. This view will be denoted as the "nutritional" hypothesis. Another alternative hypothesis is the view that smoking is a characteristic of a certain type of person or a group of people who have a poor reproductive performance because of constitutional reasons. This hypothesis will be denoted as the "constitutional" hypothesis. The latter two hypotheses have several features in common and are complementary to each other. The evidence in favour of the "causal" hypothesis is presented in great detail in the 1979 U.S. Surgeon-General's report (1) and will not be discussed in detail, but the evidence in favour of the two alternative hypotheses will be presented and their relative merits discussed.

For a valid discussion of smoking and pregnancy, the effect of factors that are already known to affect birthweight and perinatal mortality needs a brief consideration.

Factors that are known to affect fetal growth:

Biological Factors

The biological factor which shows the best correlation with the weight of the infant is the functional capacity of the placenta, which is determined mainly by the size of the placenta and the quantity and quality of the blood supply to the fetus. The size of the placenta is largely determined by the height and weight of the mother. The factors that influence the quantity of blood flow are not well understood except in pathological states such as pre-eclampsia and extensive infarctions in the placenta. There is some evidence that uterine blood flow is under hormonal control (26). The quality of the blood supply is mainly determined by the nutritional status of the mother. It is well-known that in poorer countries with nutritional deficiencies, the weight of the mother and the infant are both lower than that of those in the more prosperous countries (2). Thus, maternal stature and the nutritional status during the pregnancy appear to be important factors on theoretical grounds and are, in fact, found to be so in practice. The other biological factors which are known to affect birthweight are:-

1. The length of gestation, the shorter the gestational period, the smaller the baby,
2. Sex of the infant, males being slightly heavier than females (about 200g) after about 36 weeks of gestation, and

3. The pregnancy number, the first child being slightly lighter than later children (about 200g).

Socio-economic factors

In addition to these biological factors, it is well known that there are important socio-economic factors which are known to influence birthweight. It is not surprising that economic status could have an influence on birthweight in poor countries, probably by affecting the nutrition of the mother, but even in some prosperous western countries the weight of the infant is related to the social and educational status of the husband. Thus, social classes 1 and 2 (professional workers) have a higher birthweight, longer gestational periods, lower perinatal mortality and lower congenital deformities than those in social classes 4 and 5 (manual and unskilled workers). These socio-economic factors appear to be particularly striking in their effect on perinatal mortality in the United Kingdom. Thus, the perinatal mortality in social class 1 was 9.5 per 1,000 births and was 32.0 per 1,000 in social class 5 (3).

The striking effect of social class on perinatal mortality appears to be mainly due to the higher incidence of prematurity, low birthweight babies, and congenital abnormalities. In fact, social class has such a striking effect on the health of the population in certain countries (4) that there is a higher incidence of shorter mothers (44%) in social classes 4 and 5 compared to social classes 1 and 2 (20.5%). The number of mothers delivered before 36 weeks gestation was twice as high for classes 4 and 5 (8.4%) as that of 1 and 2 (4.2%). (See Table 1). This higher incidence of prematurity alone can account for the higher perinatal problems of the social classes 4 and 5. The socio-economic differences persist even when maternal stature has been accounted for (5). It has been shown that the incidence of prematurity is associated with the social class of the mother's father, for any given social class of the mother's husband (6). This indicated that the socio-economic status (probably nutritional status) of the mother when she was a child had a striking effect on her growth in childhood and her reproductive performance in later life. Thus, in Britain, social class appears to affect the maternal size and nutrition, and could be a very important factor which affects birthweight and perinatal mortality. In the United States also, similar socio-economic factors appear to affect perinatal mortality. Thus, black populations in general and the less educated among the whites have a higher perinatal mortality than the better educated whites (29). In a large study on perinatal mortality conducted in Canada, the hospital status of the mother, whether private or ward patient, which is probably determined by the educational status of the father, has been shown to be an important risk factor, the private patients having a much lower perinatal mortality than that of the ward patients (21).

Thus, there are several important biological and social factors which affect birthweight and perinatal mortality and these must be taken into account when considering the association between smoking and perinatal mortality.

Some Methodological considerations

Most studies on the subject of smoking and pregnancy have not taken the above factors into consideration and their conclusions, therefore, are open to question. The 1979 Surgeon-General's report has pointed out that "problems arise from lack of adjustment for differences between smokers and non-smokers in the distribution of such factors as age, parity, socioeconomic status and race when the relationship of maternal smoking to perinatal mortality is under study." (1)

The usual definition of a low-birthweight baby is one that weighed less than 2500 grams at birth. Although it is a convenient definition, it could be subject to serious errors. The gestational age, the parity, and the sex of the infant must be taken into consideration to obtain some degree of validity of fetal growth retardation. It is obvious that for a small woman weighing 100 pounds, a 2500 gram baby born at 34 weeks is not a small baby but it is for a tall woman weighing 160 pounds and delivering at 40 weeks of her pregnancy. In fact, the 2500 gram limit appears to be absurd in some situations as it has been shown that the perinatal mortality for the under 2500 gram babies increases with the increase in maternal height (7). Although it would become too cumbersome to take the mother's height and weight into consideration in defining low birthweight, at least the gestational age, parity and sex of the infant should be taken into account.

An important concept in defining fetal growth retardation was described by Gruenwald (8) and adopted in a study by Miller and co-workers (9). According to this concept, fetal growth retardation is divided into two types. One is the "long, thin baby," which is the result of wasting that occurs during a period of days prior to birth, resulting in a low ponderal index. The second type has a general decrease in growth probably extending over a period of weeks before birth, with the result that the deficits in body length and weight at birth are proportional resulting in a normal ponderal index. The second type is described as short for dates (SHFD). The infants of the first type with the low ponderal index, have large appetites and will catch up within a few months with the weight of infants with a normal birthweight. On the other hand, the SHFD infants and their siblings have a notable incidence of organic disease, suggestive of a genetic or familial pattern. It can be seen later in this paper that this type of classification of fetal growth retardation is useful in the understanding of the question of smoking and pregnancy.

The "Causal" Hypothesis

Several studies have reported a higher incidence of low birthweight babies and a higher rate of perinatal mortality to be statistically associated with maternal smoking during pregnancy. However, there are some studies in which this association is not reported. Nevertheless, this statistical association has been interpreted as having causal significance, in spite of considerable evidence against this view. It may be worthwhile to assess critically the evidence for and against this hypothesis.

If maternal smoking is the cause of fetal growth retardation and an increase in perinatal mortality, this effect of smoking should be found in all countries and in all sections of the population. However, this is not found to be true. This effect is seen only in poorer social groups, but not in the upper socio-economic groups (See Table 2). Many studies have shown that the perinatal mortality is not significantly higher in the better-educated U.S. whites, or in the upper social classes in Britain (social classes 1 and 2). On the other hand, smoking is associated with higher perinatal mortality in only certain poorer groups such as the less educated whites and blacks in the U.S.A., and the manual and unskilled workers in Britain. This lack of consistency of the so-called "effect" of smoking is against the causal hypothesis.

Fetal growth retardation is not specific to the babies of mothers who smoke in pregnancy. Other associations such as maternal stature, maternal nutrition and socio-economic status are already shown to be of causal significance.

- The strength of the reported statistical association between maternal smoking and perinatal mortality is much less than that of other factors such as previous history of perinatal loss or socio-economic status. For example social class 5 (unskilled workers) in Britain shows an excess of perinatal mortality of 400% over that of social class 1. This excess is more than ten times that which is associated with smoking (about 35%). Such a comparatively weak statistical association is not in favour of the causal hypothesis.

The claimed dose-response relationship is often quoted as evidence for the causal hypothesis. In such studies, it is obviously important to keep other factors constant, while the different doses and their response are compared. This is difficult except by statistical methods such as by the analysis of co-variance. Most of the studies which have reported a dose response relationship have not corrected for the influence of other factors. However, a study by Mayer and associates (21) have done this and claimed a small "independent" effect of smoking, with public hospital status and previous pregnancy loss showing much stronger associations with perinatal mortality. In a study of a large number of subjects (some 50,000) by Meye (22) where all the factors that are known to affect birthweight were taken into account, the difference in the mean birthweight of babies of light smokers and of heavy smokers at 37-41 weeks of gestation was not significantly different. At 39 and 40 weeks of gestation with 8454 and 10300 subjects, at each week the difference in the mean birthweight was only 2 grams and 1 gram, respectively (See Figure 1). The results of this well controlled study using large numbers of subjects should be very reliable, but do not indicate a dose-response relationship.

The temporal relationship between the change in the incidence of disease and the amount of smoking in the population has been cited as evidence in the controversy regarding smoking and certain diseases e.g., lung cancer. In the case of smoking and perinatal mortality, there is an inverse temporal relationship. Smoking has markedly increased in the last thirty years in women, whereas perinatal mortality has markedly decreased during this period in most Western countries.

In view of the foregoing, it is necessary to consider some alternative hypotheses to explain the higher incidence of fetal growth retardation and perinatal mortality reported in some groups of mothers who smoke during pregnancy.

"Nutritional" hypothesis and the role of weight gain in pregnancy.

It is well known that in poorer countries with nutritional deficiencies, the weight of the mother as well as that of the baby are lower than that of those in prosperous countries. Nutritional supplementation is known to increase the average birthweight in these poor countries. It is generally assumed that in prosperous western countries, such as the U.S.A. and Britain, there are no nutritional deficiencies. However, several studies have shown that it is not true for all sections of the population in these countries. Proteins, vitamins and trace elements which are essential for the normal development of the fetus are the most expensive of foods and could be deficient in the diet of the mothers from low-income groups (11). In a study in the U.S.A., it has been shown that mothers from a low-income group ate less protein than mothers from a high income group (12). In another study in the U.S.A. nearly a third of the mothers (53 out of 182) had a low protein-low calorie intake and had a significantly higher incidence of low birthweight babies (13). Deficiency of an extremely important trace metal, namely zinc, can occur in certain sections of the population in the U.S.A. (14). The possible importance of undernutrition as a cause of fetal growth retardation is reflected in the higher incidence of low birthweight babies in the poorer sections of the population in the U.S.A. and the U.K. For example, the upper social groups (social classes 1 and 2) have an incidence of low birthweight of 4.5% whereas for the lower social classes it is 8.2% (15). The perinatal mortality per 1000 births (3) for the upper social classes is 9.5 whereas for the lower social classes (social class 4 and 5) it is 35. In the U.S.A., the incidence of low birthweight and perinatal mortality is higher for the babies of black mothers and the less-educated whites than it is for the babies of the better-educated whites. An extremely important fact that emerges from many of the studies on smoking and pregnancy is that the excess of perinatal mortality found in mothers who smoke is not found in the prosperous groups, but only in the poorer sections of the community in the U.S.A. and the U.K.

(See Table 2). It can be seen that perinatal mortality ratios of smokers' babies to those of non-smokers' babies, varies between 1.02 to 1.13 in seven studies in which the mothers were from the upper social groups whereas for the lower social groups the mortality ratio varied from 1.39 to 2.16. These results make it abundantly clear that the socio-economic status is very important in determining the incidence of perinatal mortality.

A large number of studies have reported a higher incidence of low birthweight babies for mothers who smoke during pregnancy since Simpson in 1957 first made this observation (See ref. 1 for further references). As mentioned before, most of these have not taken into account factors which affect birthweight such as social class. In a few studies which have taken these factors into account there is no significant difference in birthweight between smokers' and non-smokers' babies (See Table 3).

The possible role of nutrition is highlighted in a recent study conducted on mothers from Bellshill Maternity Hospital (16). The birthweight was adjusted for the gestational age, parity of the mother and the sex of the infant (4). The mothers were classified according to the social class of the husband. It was found that the mean birthweight of the upper social classes 1, 2 and also 3 (professional and technical) did not show a significant difference between smokers and non-smokers whereas this difference was large and highly significant in the lower social classes namely 4 and 5 (manual and unskilled.) The incidence of small-for-gestational-age ("SFGA") infants was significantly different between smokers and non-smokers in only the lower social classes and was greater in social class 5 than in social class 4. (See Table 4). If smoking during pregnancy is the cause of fetal growth retardation, why does it occur only in certain groups of mothers and not in others? It can scarcely be argued that the so-called "effects" of smoking respect the upper social classes and pick on only the poor and underprivileged mothers.

The results from this study give an excellent example of how the same data can be interpreted in different ways, either for or against the "causal" hypothesis. The incidence of SFGA infants for all social classes as a whole was 6.4% for non-smokers and 15.2% for smokers. This difference in incidence of SFGA infants between smokers and non-smokers is statistically significant ($p < 0.01$). It would be quite easy to claim from these results that smoking was the cause of the higher incidence of SFGA infants in smokers, as has been done in many other studies. It would also be quite difficult to disprove this claim with this experimental design. However, if an attempt is made to make smoking the only variable between smokers and non-smokers by correcting, as far as possible, for factors that are known to affect birthweight, the results are no longer in favour of the causal hypothesis, and are in fact against the causal hypothesis. Thus, if smokers were classified according to the social class of the mothers' husband, entirely different incidences of low birthweight are seen in the different social classes for the same amount of smoking, and smoking mothers from the upper socio-economic groups do not show an excess of incidence of SFGA infants. If smoking does not cause fetal growth retardation in one social group, it cannot obviously be the cause in another ethnically same social group. Therefore, the association between the higher incidence of SFGA infants and smoking in the poorer social groups cannot be considered to have a causal relationship. A causal relationship might be claimed if smoking were the only variable between smokers and non-smokers. Most studies on smoking and pregnancy assume that the smoking habit is the only difference between the life-style of smokers and non-smokers.

Contrary to this assumption, there is much evidence that smokers and non-smokers differ in their personality, their attitude to life and their eating and drinking habits (23). A significant difference in the drinking habits alone could account for the higher incidence of SFGA infants in certain groups of smoking mothers. Therefore, it is entirely unjustified to claim that smoking was the cause of fetal growth retardation in any group of smokers, unless the smoking and non-smoking mothers are exactly comparable in every other way.

The gradient in the incidence of SFGA infants with decrease in socio-economic status (Fig. 4) of smoking mothers indicates that some factor such as family income, which is quantitatively associated with the social class gradient, is also quantitatively related and possibly bears a causal relationship to fetal growth retardation. Since family income could affect the nutritional status of the mother, maternal nutrition may be the cause of the higher incidence of fetal growth retardation in the poorer social groups. This view has the advantage that it will lend itself to experimental verification and has been recently verified on a group of mothers in Bellshill Maternity Hospital.

Maternal nutritional status was investigated by measuring the protein intake in the third trimester of pregnancy. Those mothers who had a low protein intake in pregnancy had a disproportionately higher incidence of SFGA infants (43%) compared to those with a normal protein intake (3.4%). See Table 6. Among mothers who smoked, those who had a normal protein intake had the same low incidence of SFGA infants as non-smokers. This indicates that nutritional deficiency, but not smoking, is a pre-requisite for the development of fetal growth retardation. Since the majority of mothers who were delivered of SFGA infants (65 out of 95, 68%) had a low protein intake, correction of this and other associated nutritional deficiencies is likely to result in a striking reduction in the incidence of low birthweight, irrespective of their smoking habits.

Further evidence in favour of the nutritional hypothesis comes from studies on maternal weight gain during pregnancy. It has been suggested that smoking mediated its so-called "effect" through maternal weight gain; Rush (17) and Davies and associates (18) have shown that the mean weekly weight gains were reflected in the infant's birthweight. Davies et al. reported that their results are consistent with the view that mothers' own weight gain has a greater effect on fetal growth with smoking having a very small "independent" effect.

Other studies (19, 20) investigating the role of weight gain during pregnancy have reported an independent "effect" of smoking when weight gain was held constant. However, when an analysis of variance was carried out (21), previous pregnancy loss and hospital status (private or public) showed a much higher degree of statistical correlation (F. ratio 97.4 and 44.2 respectively) than smoking (8.4). Miller and associates (9) have shed some light on the role of weight gain in fetal growth retardation. They found in a study of 688 non-smokers and 434 smokers that there were 62 and 67 mothers who gained less than 0.5 pound per week in the last two trimesters of pregnancy. The incidence of fetal growth retardation was five times higher among non-smoking mothers with low weight gain (less than 0.5 pound per week) than among mothers with greater weight gain. For smokers, it was three times as high among those with low weight gain than among those with greater weight gain. In the group of 42 mothers who delivered infants with fetal growth retardation, the incidence of mothers who were in the lower social classes and who smoked were significantly higher than the respective incidences among 1070 mothers who did not have infants with fetal growth retardation.

Further evidence for the importance of weight gain in pregnancy is provided by the work of Naeye (22). He studied the weight gain in successive pregnancies in groups of smokers, non-smokers and two groups of women who smoked only in either the first or the second pregnancy. Naeye found that mothers who smoked during one pregnancy but not in another had smaller infants in the pregnancy in which they smoked irrespective of the birth order, and a variety of other maternal and fetal factors that are known to affect fetal growth. He claimed, therefore, that smoking appears to have an independent effect on fetal growth. However, close examination of his data (Table 3 of his paper) shows some interesting correlations between maternal weight gain and birthweight. Since this author has recorded maternal weight gain and birthweight in both the first and second pregnancies in which smoking was the only variable, it is possible to compare the difference in maternal weight gain with the difference in birthweight between the first and second pregnancies. Such a comparison showed that the differences in weight gain between the first and second pregnancy had a highly significant correlation with the difference in birthweight between the successive pregnancies (See Figure 2). This clearly indicates that weight gain is the factor that determines birthweight, and smoking, contrary to his claims, does not have an independent effect. Since maternal nutrition has probably the most important effect on the weight gain, these results can be considered as strong evidence in favour of the nutritional hypothesis.

Constitutional Hypothesis: Smoker or Smoking?

Whether low birthweight and higher perinatal mortality is due to smoking or due to the smoker (smokers' biological and social characteristics) is a real and important question. Abundant evidence is presented above to show that smoking has no statistically significant association with low birthweight or perinatal mortality in the upper social groups, but only in the poorer, underprivileged and probably undernourished groups of mothers. In addition to these socio-economic characteristics, there are probably also genetic, biological and psychological characteristics of some smokers who will have a poor reproductive performance. It has been shown repeatedly that the poorest of the social groups have also the largest proportion of smokers. It is reported that the eating and drinking habits of the smokers are very different from those of non-smokers (23). Yerushalmy has (23) produced some good evidence in favour of the "constitutional" hypothesis. He showed that the proportion of low birthweight babies born to mothers who became smokers in later pregnancies was significantly higher than those of mothers who never smoked, and was similar to that of the babies of mothers who smoked in all their pregnancies, indicating that certain types of mothers have low birthweight babies, whether they smoked or not. In commenting upon these findings, the 1979 U.S. Surgeon-General's report (1) claimed that these results are not reliable because the mean age of the "future" smokers was 19.7 years and was significantly different from that of the non-smokers which was 22.1 years. Although Yerushalmy during his lifetime, had adequately replied to this type of criticism (24), his reply is not taken into account by the 1979 U.S. Surgeon-General's report and may therefore be worthwhile reiterating. Figure 3 drawn from Yerushalmy's data (24) is a regression of maternal age and the incidence of the percentage of low birthweight babies. It can be seen from the regression line the percentage of such babies at 19.7 years is 6.44% and for 22.1 years, it is 5.89%, a difference of 0.55% which is not significant by the Chi-Square test. On the other hand, the difference between the percentage of low birthweight babies of future smokers (9.5%) and non-smokers (5.3%) is significant at $p < 0.02$. It is also of interest that mothers who gave up smoking in later pregnancies had significantly less low birthweight babies before they gave up smoking than those who smoked in all their pregnancies. (23) Silverman (25) set out to confirm these findings, but her results were equivocal and could not either confirm or deny the findings of Yerushalmy. Further work along these lines is required to answer the question whether it is the smoking or the smoker that is the cause of low birthweight and higher perinatal mortality reported in some groups of smokers.

It should be pointed out that the higher incidence of low birthweight babies does not necessarily result in increased perinatal mortality. Table 5 shows that the perinatal mortality rate of such babies of smoking mothers is actually

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lower than those of non-smoking mothers. This paradoxical finding has never been adequately explained by the "causal" hypothesis.

Gruenwald's and Miller and associates' classification of fetal growth retardation into those with normal length for gestation with a low ponderal index (PI) and those with retardation in length and weight (SHFD) is also of relevance to the discussion of the constitutional hypothesis. The proportion of babies with a low PI was greater among smokers than among non-smokers (2.3% in non-smokers and 3.0% in smokers). If smoking causes hypoxia to the fetus and other ill effects one would not expect a higher proportion of low PI babies among smokers but just the opposite. The other category of fetal growth retardation, namely, short for gestational age (SHFD), is of much more serious pathology, suggestive of a genetic or familial pattern. Babies of smoking mothers have an excess of the latter type of growth retardation, indicating that this disorder was genetic or constitutional. As mentioned before, the mothers of such infants had a disproportionate number of women from poor social class. Furthermore, the placental weight of the mothers who had SHFD infants was significantly lower than that of normal, whereas in conditions which result in hypoxia such as altitude, placental weights are larger than normal. Under these conditions, it would be unjustified to assume that either type of fetal growth retardation was caused by smoking.

We are still far from understanding the etiology of fetal growth retardation. We do not fully understand the normal physiology of pregnancy. However, there are certain clues to the etiology of fetal growth retardation. The factors that are known to have an effect on fetal growth have been mentioned before. The quantity of blood supply to the fetus is obviously an important factor, and this appears to be affected by hormonal influences. Unconjugated free oestriol and oestradiol appear to affect uterine perfusion (26) and an increase in blood volume which occurs in pregnancy. It has been shown that maternal intravascular blood volume in pregnancy is highly correlated with fetal weight (27). As the precursors for these hormones are of fetal origin, the fetus may control its own growth potential to a large extent. Thus, the vitality of the fetus which is of genetic origin probably has the most important influence on its own growth with effects of maternal and evironmental factors superimposed upon it.

In conclusion, the evidence against the "causal" hypothesis is so considerable that alternative hypotheses are required to explain the findings of studies on smoking and pregnancy. On the other hand, the evidence for the "nutritional" and "constitutional" hypothesis is far more convincing. It is important to remain unbiased and be objective in designing studies on smoking during pregnancy and in the interpretation of their results. All the available evidence suggests that the higher incidence of fetal growth retardation in certain groups of mothers who already have a poor reproductive performance is a social problem for which smoking is not the cause but only a symptom. It is also obvious that the solution of this serious social problem, i.e. lack of proper nutrition, will be neglected if undue attention is given to smoking.

March 9, 1982

Table 1.

Incidence of Certain Characteristics in Primiparae by Husband's Social Class.

Thomson et al 1968.(4)

Husband's Social Class	Delivered at 36 weeks or earlier (per cent)	Women below 62 inches (157.5 cm.) (per cent)
I and II	4.2	20.6
IIIa	6.5	32.9
IIIb	6.5	38.1
IV and V	8.4	44.0

Table 2.

PERINATAL MORTALITY IN RELATION TO SMOKING.

	Non-Smokers number of subjects	Smokers number of subjects	Perinatal loss/1000 Non-Smokers	 Smokers	 Ratio	Reference
1. British Perinatal Mortality Survey Social Class 1 and 2 Butler et al. (1968)	11,145	4660	25.8	26.3	1.02	28
2. Boston City Hospital} Rush and Kass (1972)}White	513	892	29.2	31.4	1.08	29
3. U.S. Collaborative Perinatal Survey Niswander and Gordon (1972) White	8,521	11.369	31.4	35.5	1.13	30
4. Comstock et al. (1971) (Fathers education 9+ years)	7.646	4,641	29.2	31.4	1.12	31
5. Underwood et al. (1969)	24,865	23,629	19.7	20.8	1.05	32
6. Rantakallio (1969) (Northern Finland)	8,898	2,368	23.2	23.4	1.08	33
7. Fabia (1977) (Canada) (Maternal age 25-34)	3,192	2,962	12.6	13.2	1.05	34

Table 9.(Contd)

STUDIES WHICH HAVE SHOWN A HIGHER PERINATAL MORTALITY IN SMOKERS

		Perinatal loss/1000			
		Non-Smokers	Smokers	Ratio	Reference
1. British Perinatal Mortality Survey Social Classes 3, 4 and 5 Butler et al. (1968)		33.5	46.6	1.39	28
2. Rush and Kass (1972)	Black	28.6	54.1	1.89	29
3. Comstock et al. (1971)	White <8 years education	17.6	38.0	2.16	31
4. Fabia (1977)	White >35 years	23.6	41.7	1.81	34

TABLE 4. BIRTHWEIGHT ACCORDING TO SOCIAL CLASS AND SMOKING

(From Rao, 1979, ref. 16)

	NON-SMOKERS				SMOKERS			
Social Class	n	Birthweight g ± 1SD	S.F.D. n	S.F.D. %	n	Birthweight g ± 1SD	S.F.D. n	S.F.D. %
1.	74	3504 ± 450	4	5.40	25	3399 ± 416	-	0
2.	226	3471 ± 502	13	5.75	74	3352 ± 564	5	6.75
3.	496	3375 ± 528	32	6.45	233	3259 ± 527	18	7.27
4.	657	3452 ± 512	42	8.46	626	3178 ± 496*	95	15.17*
5.	168	3396 ± 548	14	7.14	271	3101 ± 566*	69	25.46*

* Significant difference between smokers and non-smokers $p < 0.001$

S.F.D. = Small for the gestational age, parity and sex of infant.

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TABLE 15

Perinatal deaths among all infants and among low-birthweight infants by mother's smoking status in four large studies

Investigator	Year	No. of Births	Per cent L.B.W. Infants Smoker	Per cent L.B.W. Infants Non-smoker	Perinatal deaths/1000 All Infants Smoker	Perinatal deaths/1000 All Infants Non Smoker	Perinatal deaths/1000 LBW Infants Smoker	Perinatal deaths/1000 LBW Infants Non Smoker
Underwood & Associates (34)	1967	48505	8.9	5.7	20.5	19.7	187.5	269.0
Rantakallio (34)	1969	11931	6.1	3.5	23.4	23.2	287.5	343.6
Butler & Colleagues (29)	1969	16994	9.3	5.4	44.8	32.4	268.5	284.5
Yerushalmy* (25)	1971							
White		9793	6.4	3.2	11.3	11.0	113.9	218.3
Black		3290	12.3	5.8	21.5	17.1	113.8	201.6

* Neonatal deaths only

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Table 11 Incidence of Low birthweight (LBW) according to protein intake in smokers and non-smokers

	Non-smokers	Light smokers	Medium smokers	Heavy smokers	Total
Number of pregnancies	470	189	224	127	1010
LBW in normal protein intake	16 out of 422 3.78 %	4 out of 173 2.3 %	6 out of 175 3.4 %	4 out of 95 4.2 %	30 out of 865 3.4 %
LBW in low protein intake	12 out of 47 25.5 %	9 out of 18 50 %	36 out of 48 54 %	18 out of 32 56.2 %	65 out of 145 44.8 %

LBW in all smokers: Normal protein: 14 out of 540 = 3.81 %
Low protein: 53 out of 98 = 54 %

All LBW infants with low protein: 65 out of 95 = 68 %

Light smokers (less than 10 cigarettes a day); Medium smokers (10-19 cigarettes a day)
Heavy smokers (20 or more cigarettes a day)

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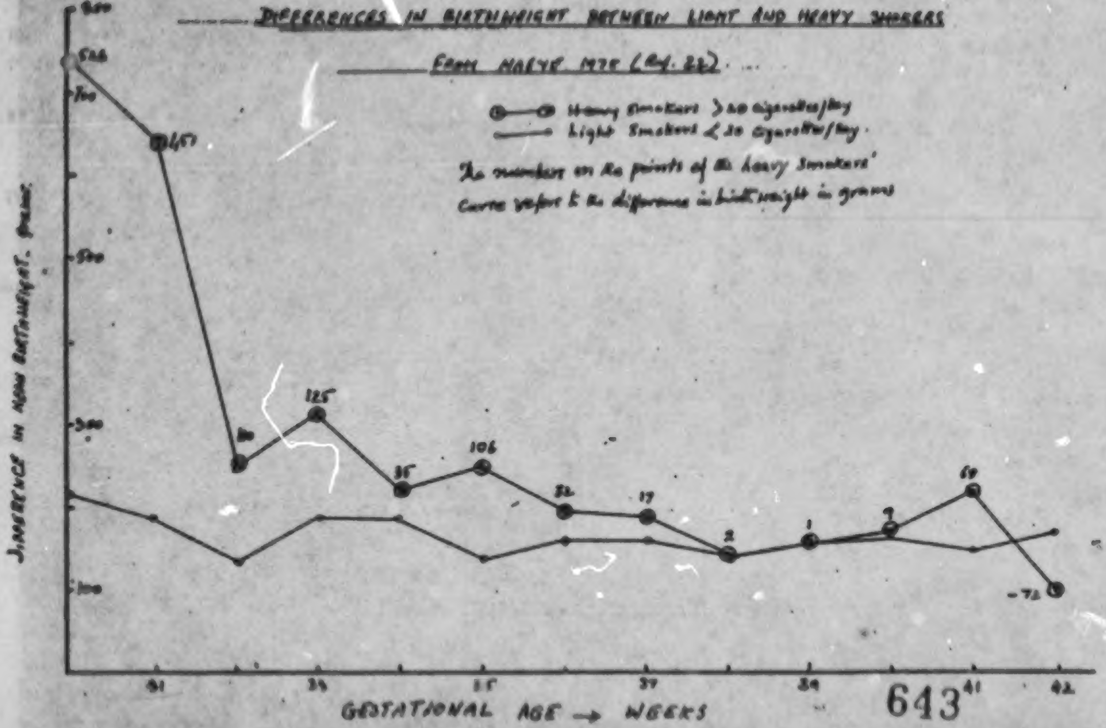
Figure 1.

DIFFERENCES IN BIRTHWEIGHT BETWEEN LIGHT AND HEAVY SMOKERS FROM NAEYE 1978 (Ref. 22) ...



The numbers on the points of the heavy smokers' curve refer to the difference in birth weight in grams

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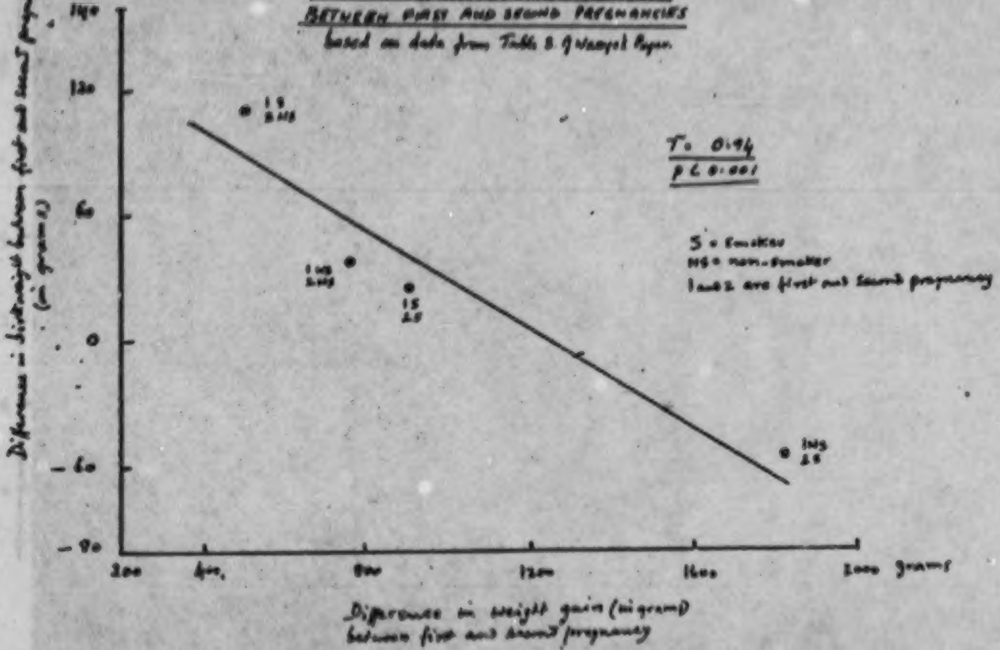
FIGURE 2

CORRELATION OF DIFFERENCES IN WEIGHT GAIN AND BIRTHWEIGHT BETWEEN FIRST AND SECOND PREGNANCIES

based on data from Table 3 of Wanyet Paper.



r = 0.94
$p < 0.001$

S = smoker
NS = non-smoker
1 and 2 are first and second pregnancy

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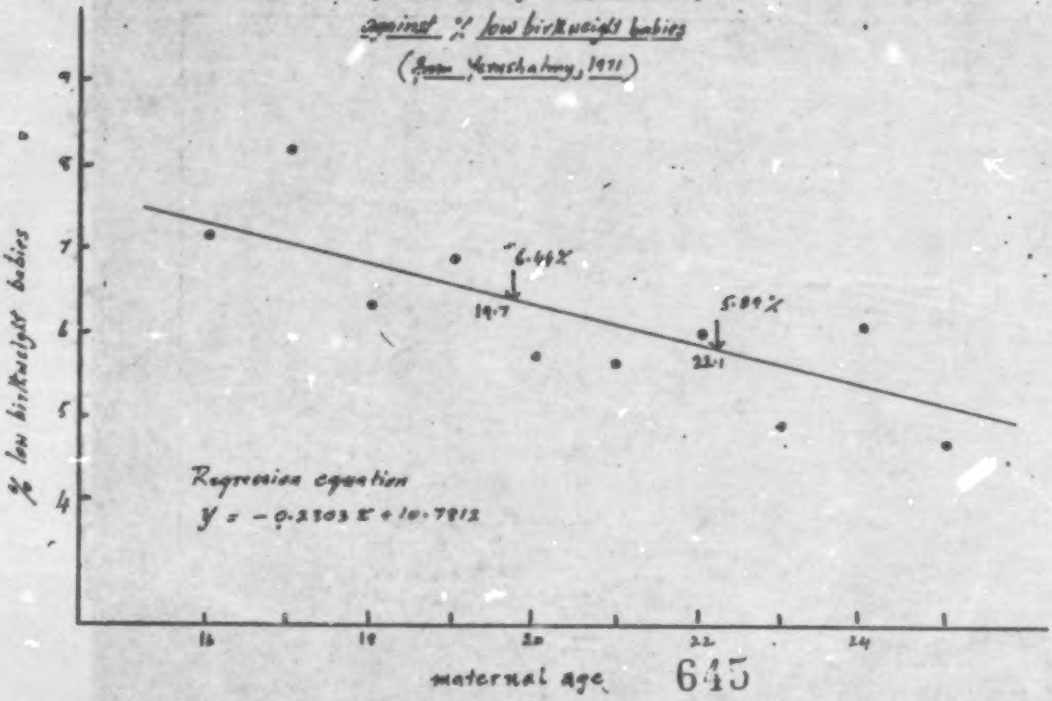
Figure 3

Regression of maternal age against % low birthweight babies

(from Yerushalmy, 1971)



Regression equation

$y = -0.2303x + 10.7812$

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I am Dr. Jay Roberts, Professor and Chairman of the Department of Pharmacology at the Medical College of Pennsylvania in Philadelphia. I also have served as a consultant for the National Research Council, NIH study section groups, and a number of private companies and laboratories. I have also been a Research Associate on the consulting staff at the Philadelphia Geriatric Center.

My current activities in the scientific research field include direct laboratory experimentation and editorial and reviewing responsibilities for scientific publications. I am on the Editorial Board for the Journal of Clinical Pharmacology, Journal of Cardiovascular Pharmacology and Journal of Gerontology.

The professional societies to which I belong include the American Society of Pharmacology and Experimental Therapeutics, the Society for Experimental Biology and Medicine and the American College of Clinical Pharmacology. I am a Fellow in the American College of Cardiology , the College of Physicians of Philadelphia and the Gerontological Society of America.

I have been asked to comment as a scientist on two bills pending before Congress, HR 4957 and S 1929. My comments will be limited to the scientific points that are raised in the proposed legislation.

My primary concern with both proposed bills is the conclusive nature of Section 2. A number of unanswered, scientific questions come to mind:

1. To what extent has the physiological and pathological changes associated with aging been taken into account?

2. Where are the data that conclusively prove one-third of heart disease deaths are attributable to smoking?

3. How much and to what degree do occupational exposures contribute to chronic disease statistics?

4. What is the relationship between smoking, aging and the physiological and psychological constitution of the individual? How do these relationships affect disease rates?

While statistical correlations are important and suggestive,the scientific conclusions reached need to be explored further. Smoking per se is not the only factor or characteristic of smokers that needs to be examined in studies. For example, the effect of stress on the cardiovascular system is well known and exposure to high levels of stress for long periods of time may have a major impact on the health of certain people. Another complicating unknown is the relationship between stress and smoking; why do people smoke and what does it say about their attempt to deal with stress?

Our animal studies involving nicotine show that it produces acute cardiovascular effects. But we need to know more about the chronic effects of nicotine in animals. Similarly, we need to know more about how nicotine is handled in the human system and how this directly relates to changes in the cardiovascular system.

My experimental work both in the cardiovascular system and in aging areas leads me to conclude that the factors involved in the development of chronic disease are complex. Despite extensive research in animals and observational work in humans, there are many unanswered questions why and how people develop diseases of the cardiovascular system. Statements in the present bills, such as, "1/3 of the deaths attributable to heart disease are associated with smoking" would seem to imply that the answers to the questions I raise are already answered and no further scientific study of this matter is warranted. But one may ask whether the diseases of the cardiovascular system in question are a natural result of the aging process or do other factors such as genetic background and lifestyle habits initiate and promote the disease?

As a scientist, I am interested in research and its role in providing reliable scientific information. I believe through further research the questions raised above could and should be answered.

Statement of Henry Rothschild, M.D., Ph.D.

Introduction

I am Henry Rothschild, a medical doctor and doctor of philosophy specializing in genetics, molecular biology and oncology, currently Professor of Medicine and Anatomy at the School of Medicine, Louisiana State University, New Orleans, Louisiana.

Since 1962 I have been engaged in research involving the genetic basis of disease and am director of the Louisiana Ethnogenetic Disease Association.

I currently serve as consultant to the Louisiana Department of Health and Human Resources and am a member of the Research Committee of the American Heart Association of Louisiana. I have published over 40 scientific articles. My Curriculum Vitae and list of publications are attached.

Discussion

Studies conducted in Louisiana have demonstrated an unusual distribution of lung cancer mortality. The lung cancer mortality in white males in Louisiana is the highest in the nation, and in particular the 27 southern preponderantly rural parishes ranked in the top 3% nationally from 1950 through 1976 and has risen since then. Studies that I have been conducting since 1975 on this population indicate that genetic factors may play a significant role in this excess mortality from lung cancer.

In our initial investigation we interviewed the next-of-kin of 284 of the 815 persons who died of lung cancer during 1971 through 1977 in the 10 southern, nonurban parishes. We found that 108 (38%) of the decedents had been employed for at least six months as sugarcane-farm workers at some time during their lives and that the relative risk estimate of lung cancer mortality for sugarcane-farm workers was more than twice that of the controls. Neither employment in other industries nor tobacco consumption could account for the elevated risk of lung cancer mortality associated with sugarcane farming.

We then began to explore the thesis expressed by many investigators including a group from the National Cancer Institute (Goffman, et al.,, 1982) that there may be "an inherited predisposition to lung cancer." Studies have shown that there are genetic factors associated with lung cancer pathogenesis. It has been found, for example, that first-degree relatives of individuals diagnosed as having lung cancer have higher rates of lung cancer that cannot be accounted for by other factors such as age, sex, birth cohort, or cigarette smoking. It was also shown that a non-smoking first-degree relative of an individual with lung cancer has about a fourfold risk of dying of lung cancer when compared to control relatives. (Tokuhata and Lillienfield, 1963, Tokuhata, 1964) A similar study reported an increased risk of lung cancer mortality in siblings of individuals with lung cancer (Fraumeni, et al., 1975)

The argument for the potential genetic regulation of tumor incidence (Bodeau et al., 1974) is supported by results of studies indicating that many mouse strains differ in their incidences for specific tumor types (Murphy, 1966). In humans, a spectrum of different genetic predispositions for each tumor implies that a distribution curve of genetic susceptibilities exists for each tumor (Reif, 1981).

Studies of distribution of cancer among different ethnic groups further support this proposition. Compared with whites, for example, blacks have a more rapidly accelerating incidence of lung cancer in the United States, Americans of Mexican and Chinese extraction (Buell et al., 1968; Fraumeni and Mason, 1974) also have a higher incidence, and American Indians (Creagan and Fraumeni, 1972) a lower incidence. Chinese in Hong Kong have an unusually high prevalence of adenocarcinoma of the lung, a relatively infrequent cell type in other areas (Belamaric, 1969).

In analyzying our data we obtained results that establish a strong genetic component for lung cancer in the Louisiana population. (Ooi et al., 1981). Of the

406 white residents of the target parishes who died of lung cancer during 1976-1979, we interviewed the next-of-kin of 328 cases. Of the 328 case families, 54 (16.5%) had first-degree relatives with lung cancer, compared with 16 (5.3%) in 304 control families. There were 61 (2.2%) lung cancers among the 2767 case first-degree relatives, compared with 18 (0.8%) in 2389 control relatives. This aggregation of lung cancer could not be attributed to differences in age at death, family size, or mortality. Six (1.8%) of the case families had two or more first-degree relatives with lung cancer. First-degree relatives of persons with lung cancer had, therefore, a lung cancer risk more than twice that of control relatives.

Comparing case and control relatives by stepwise logistic regression indicated that a person's relationship to the person with lung cancer, and the square of a person's age, are the two strongest predictors of lung cancer outcome, even after controlling for the effects of age and sex.

We are in the process of further investigating this population to attempt to ascertain whether any major genes can be isolated as being involved in the pathogenesis of lung cancer. If we can isolate such genes, it will be major step forward in unravelling the mystery of lung cancer causation.

Henry Rothschild

Henry Rothschild, M.D., Ph.D.

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I am Dr. Henry I. Russek, a practising specialist in the field of cardiovascular disease. I received my medical degree from the Royal College of Physicians & Surgeons in Edinburgh, Scotland. Formerly, I was Director of Cardiovascular Research at the U.S. Public Health Service Hospital, Staten Island, and Research and Clinical Professor in Cardiovascular Disease at New York Medical College. In addition, I served as consultant in cardiology at several other New York area hospitals.

I am a Fellow of the American College of Physicians, the American College of Cardiology, the Council on Clinical Cardiology of the American Heart Association, the American College of Chest Physicians and the International College of Angiology.

I am certified by the American Board of Internal Medicine and hold certification in the subspecialty of cardiovascular disease. I have published over one hundred and fifty scientific articles and have edited seven books concerned with diseases of the heart. Since 1968, I have been Program Chairman for the Annual Cardiovascular Graduate Symposium for the American College of Cardiology.

I have been asked to comment on proposed legislation in the current Congress (H.R. 4957 and S. 1929) which contains several statements describing cigarette smoking as a "cause" of heart disease. I would like to direct my comments to those conclusions.

For more than 30 years I have had a sustained interest in identifying the factors responsible for the high incidence of coronary heart disease among persons living in the United States. My early investigations conducted in young coronary patients and healthy controls clearly indicated that emotional stress of occupational origin was far more important statistically than high fat diet, obesity, lack of exercise or cigarette smoking in relation to prevalence of the disease.

Those early observations led to our investigation of the occurrence of coronary heart disease in professional groups in which there were significant differences in the demands and responsibilities of routine employment. In a questionnaire survey which we conducted among 25,000 persons in 20 occupational categories, a striking correlation was found between the prejudged stressfulness of occupation and the reported prevalence of coronary heart disease. Of further interest was the observation that the frequency and intensity of the smoking habit in different professional groups was directly correlated with the relative stressfulness of the occupational activity. Due to such confounding, we could not say whether smoking is independently associated with heart disease or whether it is simply related to what may be the real culprit, emotional stress.

My studies of emotional stress and clinical experience have led me to question the widely accepted view that high cholesterol, elevated blood pressure and cigarette smoking are the most important factors in the etiology of coronary heart disease. The fact is that these traditional risk factors are often absent in new cases of heart disease encountered in clinical practice. Moreover, I believe one can question seriously whether preventive measures directed against these "etiologic" influences have been successful. In this regard, our analysis of data on American physicians over a twenty-year period showed that there had been no significant change either in overall longevity or in average age at death from coronary heart disease. This observation made it evident to us that other risk factors must be involved because U.S. physicians are reported to smoke very little and no segment of our society is more aware of the potential dangers of hypercholesterolemia and hypertension.

Further insight into the etiology of heart disease was obtained during our analysis of data from large population surveys. We observed that persons who had stopped smoking of their own free will actually had a lower prevalence of coronary heart disease than persons who had never smoked. The original Framingham studies showed similar findings but no attempt was made to interpret this anomaly. Our explanation is that persons who discontinue smoking for reasons unrelated to medical necessity, fear or coercion, may possess an unusual capacity for adaptation to stress and thus a diminished vulnerability to atherogenic influences.

My clinical observations and research have led me to conclude that while smoking of cigarettes is harmful to the cardiac patient, many of the conclusions and findings about heart disease etiology need to be reexamined in a broader framework. Consistent and persuasive findings are being reported linking coronary heart disease to prolonged emotional stress, coronary-prone behavior patterns, sociocultural mobility, and stressful life events. Obviously, if cigarette smoking is involved in the pathogenesis of coronary artery disease, the relationship is far from clear and further research is needed in this complex area.

Henry I. Russek, M.D.

Statement of Bernice C. Sachs, M.D.; Seattle, Washington

After graduating from the University of Michigan Medical School "with distinction" in 1942, I took my internship and post-graduate training at Michael Reese Hospital and Medical Center, and the Institute for Pyschosomatic and Psychiatric Research and Training, in Chicago, 1942-1949. Since that time, I have practiced psychiatry and psychosomatic medicine in a pre-paid Cooperative Plan which delivers comprehensive medical care to Seattle, Washington, and its environs. The Cooperative has 325 board-certified or board-eligible physicians who serve a population of 285,000, and it owns and operates two hospitals comprising 450 beds. It is one of the first, and one of the largest, Health Maintenance Organizations (HMO) in the United States.

I am President of the Academy of Psychosomatic Medicine, Trustee of the King County Medical Society assigned to the Drug Abuse and Alcoholism Committee, member of The Cooperative's Cancer Committee, Board Member of the Education and Research Foundation of the American Society of Clinical Hypnosis, and Chairman of its Research and Grants Committee. I am past Chief of The Cooperative's Mental Health Service (1970-72, 1976-78), past President of the American Medical Women's Association, American Society of Clinical Hypnosis, American Psychiatric Association's Seattle Branch, and past

Chairman of the Cooperative's Alcohol and Drug Abuse Committee. In my medical practice, I have seen thousands of patients and given talks to and met with thousands of persons who shared my interests in community health and welfare. My curriculum vitae and bibliography are attached.

The large body of psychiatric experience demonstrates that disease is a problem of the individual as a whole, and not an autonomous disturbance localized in a particular organ. The extraordinary complexity of the activities and reactions of man leads physicians trying to understand and classify them to talk about the mind and body as if they were distinct and separate entitites. The emphasis on specialization in medical practice of past decades increases this dichotomy of thinking.

But, in every area, including smoking and health, we are dealing with a whole entity; an interrelated complex of underlying, constitutionally determined, physical and psychic patterns of behavior.

Since causes of reaction are complex, so the causes of disease are multiple. The 1964 Surgeon General's Advisory Committee on smoking and health recognized this basic proposition: "All members," the Report notes, "shared a common conception of the multiple etiology of biological processes" and "all were thoroughly aware of the fact that there are series of events in occurrences and developments in these fields, and that the end results are the net effect of many actions and counteractions."

What we cannot tell from simple statistical association--even if it is assumed that the association is real--is whether both smoking and the various diseases statistically associated with it are correlated with other hidden factors such as psychological and physiological differences which themselves exert the true causal force. Accumulated data suggest a number of reasons to believe this is so. In a presidential address to the American Cancer Society 20 years ago, Dr. Eugene Pendergass stated about cancer: "There is a distinct possibility that within one's mind is a power capable of exerting forces which can either enhance or inhibit the process of this disease."

In a statement to the Congress in 1965, when the Congress was considering proposed warnings for cigarette packages and advertising, I warned that admonitory labeling of cigarettes could well fan the fire of youthful rebelliousness, rather than deter smoking by teenagers. At that time, it was long since well-known that public health officials had branded cigarettes smoking as deleterious to health.

As I predicted, surveys following the adoption of warning statements showed a rise in smoking by children, particularly girls, fast catching up with boys. Yet, a high percentage of youngsters believe that cigarette smoking can cause lung cancer, emphysema, heart disease. Relatively more smoking students in 1971 than 1964 believe that smoking impairs health. Still, it was reported that despite the anti-smoking campaigns and programs, approximately 1-1/4-1-1/2 million adolescents were starting smoking each year.

Education to the health hazards of smoking didn't deter them. Scare techniques and preaching didn't deter them. They smoke anyway. Teenagers rebel against restriction. Teenagers feel that they should be able to do the things they want to do when adults tell them they can't do them. Teenagers want to control what happens to them.

Consider teenage sex activity, which adolescents fully know can produce venereal disease, and pregnancy, with high rates of premature deliveries and congenital anomalies. Availability of contraceptive measures to teenagers, costly and extensive educational efforts about birth control, have not decreased the number of teenage pregnancies. These pregnancies have increased.

HR 4957 purports to find the present Federal, State and private initiatives insufficient to warn the American public about the dangers of smoking. This is not the fact. The fact is that awareness does not alter behavior after a point. The people reached by warnings about smoking have been reached. The others are not going to change thei behavior, whatever the warning.

Different people smoke for different reasons, among them:

1) To cope with feelings of anxiety and stress;
2) For pleasurable relaxation;
3) To raise energy levels;
4) As a "suicidal act," not only consciously in the hope that health will be impaired, but also unconsciously as an inner-directing of aggressive behavior;
5) To control hostility and anger.

If we look at initiatives conveying health warnings that I have had a lot of experience with in the past fifty years--campaigns about alcohol, pill-popping, drugs, smoking, teenage pregnancies--we find that law (prohibition) did not abort drinking (30% or more of hospitalized patients today have alcohol-related problems); availability of contraceptives and birth control education to children did not decrease teenage pregnancies; numbers of people continue to smoke; there is more drug-taking throughout the adult and adolescent populations than ever before. The King County Medical Society Committee on Drug Abuse & Alcoholism, of which I am past Chairman, has addressed itself to campaigns for the education of physicians, as well as the general population. There has been no discernible change in behavior, unless it is in the direction of increasing tension-relieving behavior.

The way to learn about people's attitudes and reactions is to work closely, as I have, with thousands of patients. I have not seen a patient since the mid-fifties who was not aware of the assertion that they should not smoke. This information had even filtered down to children, who learned to tell their parents not to smoke in the belief that parental smoking is harmful to their parents and themselves.

But of course I see every day patients with cancer, and lung and heart disease, who have never smoked.

Medical "preventive" health is a personal problem. People are apprised of the dangers of smoking, but they have their own personal reasons for not giving up smoking. Dire medical advice coming from legislative bodies is not effective advice. Smoking is a personal medical matter, not a legal issue.

Stringent warnings have a negative effect. As The Smoking Digest, U.S. Dept. HEW, NCI, 1977 stated: "Fear-arousing messages actually increase smoking among the particularly vulnerable people and those with low self-esteem. Such messages tend to make the smoker defensive and harden his attitude and resistence. People who smoke to reduce anxiety also often smoke more after receiving a strong health threat in order to reduce their anxiety and fear."

If medicine is to teach "preventive" medicine, it must address itself to finding and promoting adaptive ways to deal with stress. The 1964 Surgeon General's report stated:

> "Stress seems to be related to smoking and there is evidence that the experience of stressful situations contributes to the beginning of the habit, to its continuation and to the numbers of cigarettes consumed."

The Report further stated:

> "Existence of an association between stress and tensions on the one hand and smoking behavior on the other can probably be accepted with a reasonable degree of confidence."

From the research material in the smoking field we could conclude that the same stress that stimulates heavy smoking may be the stress that precipitates the complex cancer process or produces the coronary-prone individual.

It is up to the medical profession to reduce stress in the population -- and not by fiats or mandates that engender guilt, anger and resistance, which in turn create more stress and can promote the very diseases they are trying to prevent.

It is costly and inappropriate for Congress to busy itself in this area. "Good health" and "good behavior" cannot be legislated. It is physicians who should and do address themselves to the person -- to the person's constitutional diathesis and self-motivation, and to techniques for reducing the person's stress with its deleterious physiological reactions and maladaptive responses.

Bernice C. Sachs, M.D.
March 2, 1982

BERNICE COHEN SACHS, M.D.

MENTAL HEALTH SERVICE

GROUP HEALTH COOPERATIVE OF PUGET SOUND

200 - 15th Avenue East (206)-326-7050

Seattle, Washington 98102

CURRICULUM VITAE and BIBLIOGRAPHY 1982

1918 - Born September 16, Passaic, New Jersey

1939 - B.A. Cum Laude, University of Michigan

1942 - M.D. "with distinction," University of Michigan Medical School

1942-1949 - Internship and post-graduate medical training in Chicago at Michael Reese Hospital and Medical Center and the Institute for Psychosomatic and Psychiatric Research and Training at Michael Reese Hospital and Medical Center.

1949 - Practice of Psychiatry and Psychosomatic Medicine at Group Health Cooperative of Puget Sound; Chief of Mental Health Service, Group Health Medical Center 1970-72, 1976-78

HOSPITAL STAFF:

Group Health Medical Center
St. Francis Xavier Cabrini Psychiatry Service

PROFESSIONAL SOCIETIES:

- American Medical Association
- Washington State Medical Society
  - Task Force on National Health Insurance
  - King County Medical Society Alternate Delegate
- King County Medical Society, Media Relations Committee 1973-76
  - Mental Health Committee 1970-73
  - Chairman, Alcohol and Drug Abuse 1974-81
  - Trustee 1982-84 and Delegate to the Washington State Medical Association
- Fellow of Academy of Psychosomatic Medicine, Credentials Comm., 1980
  - Executive Council 1973-76, Secretary 1976-77, Executive Council 1978-81, President-Elect 1981 - President 1982
- Washington Academy of Clinical Hypnosis, President 1970-74; 1975-77
  - Honorary President 1978-
- Society for Clinical and Experimental Hypnosis (Fellow)

PROFESSIONAL SOCIETIES: (cont.)

Puget Sound Group Psychotherapy Association
Fellow of American Society of Clinical Hypnosis, Component Section
    Comm. 1970-71, National Treasurer, 1973-76, Workshop Faculty 1973-77,
    First Vice-President 1976-77, President 1977, Newsletter Editor 1978-79
Fellow of ASCH - Research and Education Foundation
    Board of Trustees, Chairman of Grants, 1981-4
Seattle Medical Women's Association, President 1959-1964
American Medical Women's Association, President 1965

Washington State Delegate	1959-61
National Recording Secretary	1959-60
2nd Vice President	1960-61
Vice President	1961-63
President-Elect	1964-65
President	1965
Councilor: Growth & Development	1966-68
Nat. P & PR Chairman	1973-75

Pan-American Medical Women's Alliance

Corresponding Secretary	1960-62
Vice-President of USA	1962-64

Medical Women's International Association
Puget Sound Association of Phi Beta Kappa
A.M.W.A. Sponsor of the BERNICE C. SACHS JUNIOR BRANCH OF UNIVERSITY OF WASHINGTON SCHOOL OF MEDICINE
Women's Medical College of Pennsylvania (Board Member, ex-officio)
American Association for the Advancement of Science
Aerospace Medical Association
American Psychiatric Association - President-Elect, Seattle Chapter 1975-76
    President, Seattle Chapter 1976-77
Council of Medical Administrators
International Platform Association
American Geriatrics Society, Founding Fellow, West Division, 1973
American Psychosomatic Society
American Psychiatric Association - Observer - Consultant to Council of
    International Affairs 1978-79
    Observer - Consultant 1978-79
    Committee on Professional Liability 1980-86
    "   " Loss Control Review 1980-86

HONOR SCHOLARSHIP SOCIETIES:

Alpha Lambda Delta (Freshman Honorary)
Iota Sigma Pi (Chemical Honorary)
National Forensic League (Distinction in Debating)
Phi Beta Kappa (Elected to National Honorary in Junior Year)
Phi Kappa Phi (Senior Honorary)
Alpha Omega Alpha (Elected to National Medical Honorary in Junior Year)

COMMUNITY ORGANIZATIONS:

Board of Directors: Health & Welfare Council 1957-1960
Past Presidents' Assembly 1961-1962
Seattle-King County Safety Council 1960-1970

Governor's Advisory Committee on Youth Opportunities 1962-1965

Group Health Credit Union - Board of Directors - 1978-84 (Secretary)

NATIONAL ORGANIZATIONS:

Soroptomist
Women's University Club of Seattle
American Association of University Women
National Resource Personnel Board of the Intercollegiate Association of Women Students
Women's Conference Committee of National Safety Council
Mortar Board

HONORS:

1955 - Gold Key for "Most Outstanding Citizen" at 20th High School Reunion

1964 - "Bernice C. Sachs Junior Branch of American Medical Women's Association" organized by the University of Washington Medical Women Students

1965 - Theta Sigma Phi (National Honorary Journalism Society)
Special Honors Award for "Woman of Achievement"

1966 - "Medical Woman of the Year", A.M.W.A. presentation, Washington, D.C.
"In recognition of outstanding contribution to her profession, her community and to her fellow human beings"

1967 - "Myrtle Wreath Award" - National Hadassah "In recognition of her continuing contribution to humanity in the field of medicine"

1969 - Honorary Member of Mortar Board, National Senior Honorary
"because her career exemplifies the Mortar Board goals of leadership, scholarship and service," Tolo Chapter, University of Washington

1975 - "SALUTE AWARD"
"For an outstanding contribution to the Convention and Visitor Industry of Seattle and King County"

1979 - Elizabeth Blackwell Medal in honor of outstanding contribution to medicine and advancing the cause of women in medicine

1980 - Presidential Merit Award
"In sincere appreciation of her dynamic and caring service to the American Society of Clinical Hypnosis"

KNOWLEDGED IN:

Marquis - Who's Who in Washington - Century 21 Edition
Who's Who in the West
Who's Who of American Women
Who's Who in America
Who's Who in the World
Dictionary of International Biography
International Scholars Director
National Register of Prominent Americans and International Notables 1974-75
Notable Americans of the Bicentennial Era

T.V. Appearances:

National: December 1965, GIRL TALK
January 1966, Johnny Carson: TONIGHT SHOW

Local: October 1966 - KIRO "Eye of Seattle"
February 1967 -Judith Lane Show
October 1967 - KING - Community Services Program
February 1971 - KING, KOMO, KIRO - "Psychological Rights and Responsibilities of Women"
September 1971 - KCTS - Al Page - Interview: "Menopause - the Pause that Refreshes"
March 1972 - KOMO "Viewpoint"
May 1972 - KING "Telescope" - Jean Enerson, Howard Hall "Middlescent Woman"
February 1975 - KUOW FM - Univeristy of Washington, "Psychosomatic Medicine"
June 1976 - KING "Seattle Today" - "Psychosomatic Medicine"
February 1977 - KING "Seattle Today" - "Aging Gracefully"
March 1977 - KING "It's About Time"
March 1979 - KING "Today Show" - "Women Over Forty"
March 1980 - KING "Today Show" - "Women's Issues in Mental Health"

SPEECHES:

International: Puerto Rico, Columbia, South America, Tokyo, Canada, Austria, Australia, Israel, Rio de Janeiro

National: Chicago, Detroit, Rochester, New York, Augusta, Cleveland, New Orleans, Gulfport, Los Angeles, Santa Cruz, San Francisco, Portland, Olympia, Minneapolis, Phoenix, Philadelphia, Walla Walla, Sea Island, Ga., San Jose, Sarasota, Boston, Honolulu, Anaheim, New Jersey

FAMILY: Married in 1941 to Surgeon; two children, three grandchildren

CONSULTANT:

Chicago Board of Public Health 1965-1966
Washington, D.C. June 1966 - Medicare Conference
Invited by President Johnson
Washington, D.C. January 1968 - Conference on Meeting Medical Manpower
Needs: Optimum Utilization of Women Doctors, Invited by U.S. Dept.
of Labor, Women's Division

FACULTY:

Hypnosis Workshops of American Society of Clinical Hypnosis and
Education and Research Foundation
Hypnosis in Clinical Practice, sponsored by University of Pennsylvania,
Department of Psychiatry and Institute of Pennsylvania Hospital 1978-1980
Group Health Prevention Rounds, 1980-

PUBLICATIONS:

"Psychosomatic Medicine", Journal of American Medical Women's Association, March 1961

"Psychosomatic Aspects of Accidents", Industrial Medicine and Surgery, 31:12, 525-532, December 1962

"Psychosomatic Aspects of Accidents", British Columbia Journal of Medicine, 6(5), 188-192, May 1964

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CONCERNING THE "COMPREHENSIVE SMOKING PREVENTION ACT OF 1982"

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I am Professor of Chemistry at the University of California, San Diego. I hold a Ph.D. degree in chemistry from the University of Munich and am the president and founder of the International Association of Bioinorganic Scientists. I am a member of several scientific societies, including the American Chemical Society, the Association of Clinical Scientists, and the American Public Health Association. I am the author of approximately 200 research publications and have edited 2 books.

My main research interests are in cancer-prevention, cancer epidemiology, trace minerals in human and animal nutrition, and various fields of experimental chemistry. I have done pioneering work on the prevention of cancer by the essential trace mineral selenium and in 1978 received a special award from the Santa Clara Section of the American Cancer Society.

As a chemist, cancer researcher and American Citizen I wish to comment upon the "Comprehensive Smoking Prevention Act of 1982" recently introduced into U.S. Congress. In this Act, it is stated, among other things, that "smoking is the number one cause of lung cancer in the United States". In my opinion, what role, if any, smoking plays in the causation of cancer, including lung cancer, has still to be determined.

Those who claim smoking causes cancer rely upon the reported statistical association and ignore the inconsistencies of the smoking causation theory in the scientific literature. For example, to date, no one has ever been able to produce lung cancer in laboratory animals through exposure to fresh, whole cigarette smoke. Moreover, the vast majority of smokers never develop lung cancer and there are serious inconsistencies in the epidemiological evidence and dose-response relationships. For example, a Japanese male smoking 50 cigarettes per day has a lower risk of dying from lung cancer than a British smoking doctor smoking only 1-14 cigarettes per day.

In addition, no ingredient or combination of ingredients, as found in tobacco smoke, has been shown to cause human lung cancer. Tobacco smoke is a very complex mixture of thousands of ingredients. Some of these ingredients, in isolation, have been reported to be carcinogenic in test animals. However, this does not mean that tobacco smoke is harmful to humans. Compounds that are carcinogenic in test animals, when applied in isolation, have been found to be anti-carcinogenic when applied in combination with each other. Since it is probable that the many hundreds of compounds present in smoke interact with each other, it is highly artificial to focus upon the effects of any one ingredient or combination of ingredients in isolation from the others. It has long been known that certain smoke constituents act as anti-carcinogens in test animals. For example, tobacco belongs to the selenium accumulating group of plants and selenium has been shown to possess anti-carcinogenic properties. Also, constituents of cigarette smoke previously thought to be lacking altogether in carcinogenic activity have recently been found to be anti-carcinogenic when applied with true carcinogens in test animals.

Cancer is an extremely complex, multifactorial disease. Studies indicate that many factors, other than smoking, are statistically associated with cancer. Some of these factors are familial predisposition, exposure to tumor viruses and other biological causing agents, exposure to ionizing radiation and industrial carcinogens, diet, exogenous environmental factors and stress.

Further, studies indicate that a number of agents normally present in foods may have activating effects on tumor viruses and that there is an association between lung cancer mortalities and other variables of life-style and diet. For example, the lung cancer mortalities are correlated with the per capita intakes of sugar and milk, and with the consumption of seafoods. It also has been repeatedly suggested and recently reaffirmed that the consumption of diets rich in pro-vitamin A (carotene) may have lung cancer protecting effects.

The "findings" in the "Comprehensive Smoking Prevention Act" have not been proven. Moreover, passage of the Act will divert attention from other etiologic leads to the disvantage of the American Public and the progress of the health sciences.

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is professor of chemistry at the University of California, San Diego. He received his Ph.D. Degree from the University of Munich, Germany, in 1956. An inorganic chemist by training he has become a major exponent in the new field of bioinorganic chemistry. He is internationally known for his work on the role of selenium and other trace elements in nutrition and carcinogenesis. He is also a leading specialist in the areas of catalysis, biocatalysis, organometallic and coordination chemistry. Dr.Schrauzer is the founder and president of the International Association of Bioinorganic Scientists, Editor-in-chief and founder of the journal "Biological Trace Element Research" and former editor and founder of the journal "Bioinorganic Chemistry". He is the author of more than 200 research papers and has edited two books. He was born in 1932 and received his academic training at the University of Munich. He immigrated to the United States in 1964, is a Naturalized U.S. Citizen and has been at the University of California, San Diego, since 1966. Schrauzer is a member of the American Chemical Society, the Association of Clinical Scientists, the American College of Toxicology and an Affiliate Fellow of the American College of Nutrition.

I am Dr. Carl C. Seltzer, Honorary Research Associate at the Peabody Museum, Harvard University and Professor of Nutrition, Tufts University. I was formerly Senior Research Associate at the Harvard University School of Public Health. My work in the smoking and health area is extensive and I have published over thirty-five (35) articles since 1964 on the subject, many dealing with the relationship between smoking and coronary heart disease (CHD). I was a consultant to the Surgeon General's Advisory Committee on Smoking and Health (contributed a section to the 1964 Report) and am presently a Fellow of the Council on Epidemiology of the American Heart Association. My investigative work in the heart disease field includes participation in studies involving the Framingham Heart Study, The American Medical Association, the Veterans Administration and the Kaiser-Permanente Foundation.

H.R. 4957 proposes that the following label statement appear on cigarette packages: "Warning: Cigarette smoking is a major cause of Heart Disease." This proposal is apparently based on the claim, put forth as a Congressional "finding" in the first part of the bill, that one third of deaths from CHD are "attributable to smoking."

These bald statements are not based on consistent, valid, demonstrable scientific evidence, and are without established proof. Apparently, they are based on the views of a succession of Surgeon Generals subsequent to the 1964 Report, on the statements of the American Heart Association, the Framingham Heart Study and others.

The primary basis for these views can best be characterized by statements from the 1981 Surgeon General's Report and by the Framingham Heart Study. The 1981 Report stated: "The effect of smoking on CHD risk fulfills many epidemiological criteria for a causal association: powerful, independent, dose related, and reversible." Kannel of the Framingham Heart Study stated: "Causal inferences are supported by the fact that the relationship is strong, consistent, demonstrated prospectively, independent of associated risk factors, and can be explained by the known effects of cigarette smoking on the cardiovascular apparatus. Finally, it has been shown that those who quit smoking have only half the risk of those who continue to smoke. Epidemiologic data have . . . tended to indicate an independent, transient, noncumulative, reversible, triggering effect of cigarette smoking." (JAMA, 1981)

Accordingly, the main evidence advanced for a causal relationship between cigarette smoking and CHD is essentially epidemiologic, to the effect that the association is (1) strong, (2) consistent, (3) dose-related, (4) independent of associated risk factors, and (5) reversible by stopping smoking. (6) In addition, the association is said to be explained by known effects of cigarette smoking on the cardiovascular system. Let us examine these claims in turn.

Claim 1. Is the relationship strong? The 1964 Surgeon General's Report stated that in the U.S. the mortality ratio of smokers to nonsmokers is 1.7. But, this is not a strong relationship, according to Professor Hutchison of the Harvard School of Public Health. (Bull NY

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Acad Med, 1968) This independent observer feels that a mortality ratio of two or less is actually indicative of a weak relationship which may possibly be explained by confounding factors.

Can the relationship be characterized as "strong" when the 1979 Surgeon General's Report, although discussing smoking and health studies throughout the world, limits its conclusion to "men and women in the United States," and Kannel of the Framingham Study talks about the excess CHD death rates of smokers "in most of Western societies"? (AHJ, 1981) If the relationship between smokers and CHD is "strong," why is it not present in all Western societies or in non-Western societies?

Claim 2. Is the relationship consistent? Since no significant statistical associations between smoking and CHD have been reported in Finland, the Netherlands, Italy, Greece, Yugoslavia, Japan and Puerto Rico, it is clearly incorrect to claim that the relationship is consistent.

The relationship is not even consistent in the Framingham Study. It appeared to be consistent in initial analyses, but when standard risk factors and such psychological factors as Type A personality were controlled, it was found that "cigarettes smoked per day" were not predictive of CHD and myocardial infarction incidence in men, or of CHD and angina pectoris incidence in women. (Am J Epidemiol, 1980)

Can the relationship be characterized as consistent when the 1979 Surgeon General's Report concluded that "the predictive risk factor association of smoking with the incidence of angina pectoris is not clear"? The Framingham Heart Study also reports that "the relationship to angina pectoris is modest, if it exists at all." (JAMA, 1981)

Is the relationship consistent when the relative risk progressively declines with increasing age? Seltzer demonstrated and Kannel noted that the benefits of quitting smoking do not extend beyond the age of 65 as regards heart attacks. (Am J Med Sci, 1978; JAMA, 1981) Furthermore, the 20 year follow-up data of the Framingham study show no excess incidence of CHD in men or women 55 years and over. (Framingham data made available to Seltzer as consultant.)

Can the relationship be characterized as consistent when the Framingham Heart Study reports an absence of a relationship of smoking and CHD in women?

Claim 3. Is the relationship dose-related? CHD rates do not consistently show a rising gradient in relation to an increased amount of cigarette smoking. (JAMA, 1968) In some studies, the gradient is actually reversed. (JAMA, 1968) I found that data from the Framingham Study do not show a consistent rise in CHD risk with increasing amounts of cigarette smoking because of the "heterogenous" character of its "none" category, a deceptive procedure. The "none" category in Framingham consists of a combination of never smokers, ex-smokers, pipe and cigar smokers. A Framingham Study report confirmed that "there is no distinct dose-response relation [of CHD and death rates] with increasing amounts of cigarette smoking" over an 18-year surveillance period. (Lancet, 1974)

There is no significant dose-response relationship between smoking and CHD in studies of Finland, the Netherlands, Greece, Italy, Yugoslavia and Japan. (Circulation, 1970)

No claims have been made lately about the alleged association of CHD with duration of cigarette smoking. In the combined Albany and Framingham Heart Studies (NEJM, 1962), no association was found between duration of heavy cigarette smoking and risk of myocardial infarction. In a study of Canadian veterans, no consistent gradient occurred between CHD mortality and years of cigarette smoking. (JAMA, 1968) In Kahn's analysis of Dorn's U.S. veterans data, there was also no significant relationship between duration of cigarette smoking and risk of death from CHD. (JAMA, 1968)

Claim 4. Is the relationship independant of associated risk factors? Smoking has been found to be associated with CHD independently of associated risk factors in some studies when only cholesterol and blood pressure were considered. But this generally has not been the case when other risk factors were investigated, such as Type A personality, and other psychological factors (as seen above in the Framingham Study).

Claim 5. Does stopping smoking reduce the risk of CHD? This claim is based on studies from some Western countries which report that CHD rates of ex-smokers are lower than those of continuing smokers. The 1974 Lancet article on the Framingham study is the most widely quoted source for claiming that the rate of CHD among those who stop smoking is reduced to one-half that of continuing smokers. When I was provided with the CHD rates of Framingham "never smokers" (which had been omitted in the article), however, I was able to show that the CHD rates of ex-smokers were far below those of "never" smokers," while the rates of "never smokers" were not far below those of continuing smokers. (Seltzer letter to Lancet, 1977)

Additional evidence about the fallacy of the claim that stopping smoking reduces the rate of CHD comes from two other sources. The first is a study challenging the assumption by epidemiologists that ex-smokers and continuing smokers are alike in all pertinent characteristics, except for their smoking habits. If this assumption is false, which I believe it to be, then comparisons of the two groups would be biased as to their eventual CHD rates. Friedman, Seltzer, and others have shown that ex-smokers, when they still smoked, were not a representative sample of the population of continuing smokers. The data showed, in fact, that ex-smokers were at lower heart disease risk (and healthier) before they stopped smoking, than those persons who continued to smoke. (*J Chron Dis*, 1979) This would appear to account for the fact that ex-smokers have lower CHD rate outcomes than continuing smokers, not because they stopped smoking but because they are different kinds of persons to begin with through self-selection.

In a subsequent article published in the *NEJM*, Friedman and others tried to negate the results of that paper by applying theoretical correction techniques through multivariate analysis to the lower CHD-related characteristics of the ex-smokers. They claimed that the lower CHD-related characteristics actually made little difference, that ex-smokers still showed lower CHD outcomes than continuing smokers. Although this paper was criticized by Burch and Seltzer (Burch and Seltzer letters to *NEJM*, 1981), Friedman evaded virtually every criticism in his reply. (Friedman letter to *NEJM*, 1981) Friedman did not explain how he could accept the value of CHD-related characteristics (risk factors) in one paper and deny their importance in a second paper. Nor did Friedman explain why the mortality data in his later paper showed that never smokers exhibited higher CHD rates than the ex-smokers (the same anomaly as that found in the Framingham Study). Does this mean that it is better to begin smoking and then to quit than never to have smoked at all?

The second source of evidence which points to the fallacy of the claim that stopping smoking reduces the risk of CHD is the first large-scale "intervention" trial of the effect of smoking cessation in middle-aged London civil servants at high risk of cardiovascular disease. (*J Epidemiol Comm Health*, 1978) After nearly eight years of surveillance, the group subject to intervention for smoking cessation showed no improvement in its rate of mortality over the group which was not urged to stop smoking. Thus, the alleged reversibility of the risk to the smoker's life was not demonstrated.

This result is consistent with a more recent intervention trial from Norway which examined the effects of both smoking and cholesterol reduction. (*Lancet*, 1981) Although reductions in dietary cholesterol produced a statisticially significant decrease in CHD, reductions in smoking did not. This led the authors to conclude that past studies may have "overestimated the decline in risk of CHD" following reduced cigarette consumption.

Claim 6. Kannel of the Framingham Study claims that: "causal inferences . . . can be explained by the known effects of cigarette smoking on the cardiovascular apparatus." (*JAMA*, 1981) There is no satisfactory evidence for this claim, and, in fact, there are contradictions. The cold reality is that the mechanisms by which tobacco smoking allegedly causes CHD have not been established. Much of the evidence to support this statement is contained in Seltzer's article, "Smoking and Coronary Heart Disease: What Are We to Believe," published in the *American Heart Journal* as an editorial in September, 1980. This editorial discusses those statements of the 1979 Surgeon General's Report which illustrate the absence of firm evidence of mechanisms by which smoking affects CHD, such as: (1) little is known about the mechanisms by which smoking enhances atherogenesis, (2) further research is needed to show the mechanisms of sudden death and its precursor states, (3) the data on the effect of smoking on blood lipids are not very uniform, (4) the association between cholesterol and smoking is minimized, (5) the acute and transient effects of smoking are to increase heart rate and blood pressure to a minor degree, (6) smoking is not a risk factor for hypertension, (7) the association of smoking with necropsy manifestations of atherosclerosis, shown principally by Strong's group in New Orleans, now has been contradicted by a study by Holme, Strong and others who found that smoking did not show a significant association with coronary artery lesions. (*Arteriosclerosis*, 1981)

More evidence comes from other sources. An editorial in the August 1980 *British Medical Journal* states that the mechanism by which smoking affects coronary heart disease is unknown. The American Heart Association *Heartbook* states: "The mechanisms by which cigarette smoking is associated with higher rates of coronary heart disease are not yet fully understood." The 1981 Surgeon General's Report states: "Estimation of the impact of varying cigarettes on coronary heart disease risk is difficult, because the exact etiologic agent(s) have not been identified." Thus, the bill's proposal to label cigarette packages with levels of tar, nicotine, and carbon monoxide is not justified on scientific grounds. The Surgeon General admits that it has not been clearly demonstrated that these factors are prime etiologic agents in the causation of increased CHD in smokers.

This is also shown by the conclusion of the 1981 Report that despite radical changes in the composition of cigarettes over the last 10 to 15 years (filter, tar, nicotine), there was no clearly demonstrated effect on cardiovascular disease. This surprising result is clearly consistent with the proposition that amounts of tar, nicotine, and carbon monoxide in cigarette smoke have no demonstrable effects on CHD rates, and that these components of tobacco smoke are not proven etiologic agents.

Since most cardiologists believe that atherosclerosis is the most important factor in coronary heart disease development, they claim that smoking enhances atherosclerosis, despite the lack of definitive evidence. On the other hand, the Framingham Heart Study has disavowed the atherosclerotic argument and claimed that the effect of smoking on the heart is acute (not progressive), and disappears promptly when smoking is stopped. These are contradictory claims and illustrate the lack of definitive knowledge in this field.

It is clear from the above that extensive research data do not support the Congressional finding in H.R. 4957 that a major proportion of the CHD deaths in this country are attributable to smoking. In turn, the bill's proposed warning, "Cigarette smoking is a major cause of Heart Disease," is not scientifically valid.

In every instance, an examination of the claims made against cigarette smoking and CHD shows that they are either wrong, inconsistent, selective, unsubstantiated, or, in many respects, contrary to the statements of the Surgeon General, the American Heart Association and the Framingham Heart Study.

Signed Carl C Seltzer

Date 2/25/82

STATEMENT OF SHELDON C. SOMMERS, M.D.

I am Sheldon C. Sommers, M.D., a physician specializing in pathology, currently Clinical Professor of Pathology at Columbia University College of Physicians & Surgeons, New York, N.Y., and University of Southern California School of Medicine, Los Angeles, California. Also I am consultant in pathology, Lenox Hill Hospital, New York; Chairman, New York State Mental Hygiene Medical Review Board; and President-Elect, Arthur Purdy Stout Society of Surgical Pathologists. I am past president of the New England Pathological Society and New York Pathological Society.

Since 1936, except for World War II years, I have been engaged in medical research with particular reference to cancer, endocrine and gastrointestinal diseases, with over 300 publications -- about 10 percent dealing with lung and lung cancer, and also some on pancreatic cancer. I am coeditor of Pathology Annual and Diagnostic Gynecology & Obstetrics and serve on the editorial boards of five other medical journals. My curriculum vitae and publication list are attached.

For the past six months, I have served as Scientific Director, Council for Tobacco Research USA, Inc. This is a funding agency for biomedical research in the area of smoking and health, funded by tobacco manufacturers. The budget for research grants in 1982 is 7 million dollars. Applications are acted upon by a Scientific Advisory Board, and those approved with a favorable rating are funded for up to three years with an opportunity for continuation grants thereafter. The Council for Tobacco Research exerts no influence upon the grantees, who may freely publish what they find as they choose. About eighty active grants now exist in the U.S. and abroad.

My appearance at this hearing is voluntary, and the opinions expressed are personal, not representing those of any organization. They are the result of over 45 years of study, investigation and practice in the field of pathology and clinical-pathologic correlations of diseases, some of which have been attributed to cigarette smoking.

In the field of science, knowledge is gained through experiment and interpretation, the scientific method. A theory is proposed. Thereafter, experiments confirm or refute it. If the latter, a new theory is developed. It is a continuous evolutionary process, and needs a critical and open mind. One must be constantly alert for surprises, as Lewis Thomas wrote.

Lung cancer is high in the list of statements in Bill HR 4957 attributing diseases to cigarette smoking. There are two general methods of investigating the cause of cancers in humans or animals. One is the biomedical method. Cause might be defined biologically as something both necessary and sufficient to cause a condition. Cigarette smoking is not a necessary factor in human lung cancer, which existed for centuries in radium miners before cigarettes were invented. Lung cancer accompanies scarring processes in the lung due to TB, connective tissue diseases and various other abnormalities in nonsmokers.

Also, cigarette smoking fails to meet the causal criterion of being sufficient. The fact is that the vast majority of smokers, more than 90% of even heavy smokers, do not develop lung cancer. Hence cigarette smoking is neither necessary nor sufficient in the development of human lung cancer, and by the biological definition is not the cause.

Like many other diseases of older age, lung cancer appears to be multifactorial, which means the disease is associated with many things, in addition to smoking. Practically all active researchers now agree on this point. These include:

(1) Heredity. Families with lung cancer are known. Other families have decreased lung function.

(2) Sex and Race. Men have three to six times more lung cancer than women. Blacks and orientals differ from whites in incidence.

(3) Urban. Certain urban areas have high lung cancer rates which cannot be accounted for by smoking. These areas are characterized by severe winter weather inversion patterns with high air pollution.

(4) Occupation. As already noted, some twenty occupations involve increased risk. Some workers smoke while others do not.

(5) Immune competence. Individuals with demonstrably reduced immunity have developed lung cancer. This may explain some family cases, and the increase of cancer with age.

(6) Hormones. Adrenal and sex hormones accelerate some metabolic processes leading to human lung cancer.

(7) Aging. The mean age of lung cancer diagnosis has been reported as about 67 years, and said to be rising to older ages in some populations.

Currently, researchers do not know which, if any, of these or other factors play a role in the causation of lung cancer.

The other approach to cancer causation investigation is epidemiologic and statistical.

Edpidemiologic statistics involves an experimental group and a control group. For a valid comparison, the groups must be alike as nearly as possible in all respects except for the item being investigated. In studies of cigarette smoking, matching smokers and nonsmokers by sex and age was achieved, and it has been assumed that in all other respects the two groups were comparable. This is not true, since in body build, extroversion-introversion, marital history, alcohol use, use of nonprescription medications, police records, military records and other aspects, cigarette smokers are demonstrably different from nonsmokers.

The fallacy of a one-to-one comparison of smokers and nonsmokers with respect to mortality was pointed out in a monograph by Rose and Bell in 1971. They studied predictors of longevity in war veterans from Boston, re-examined at intervals. One-on-one comparisons placed cigarette smoking #1 as a predictor of early death, like many other studies. Multifactorial statistical analysis dropped smoking to somewhere below #30 as a predictor, and "dissatisfaction with job" became #1. The lesson is that in the present relatively undeveloped state of epidemiology to beware of facile and sweeping conclusions.

Where do the data on deaths from lung cancer and other conditions come from? The diagnoses are largely from death certificates. Most are not supported by autopsy examinations. A death certificate is an authorization for burial not requiring permission of the coroner or medical examiner. It is a legal but not a scientific document. When death certificate diagnoses are compared to autopsy diagnoses of lung cancer, errors in the death certificates have been found to range from 30 to 60 percent. New studies in the U.S. and other countries in the past two years again show this large error.

The basic data for lung cancer incidence thus are of poor quality and uncertain verification. The problem is getting worse since the ACS dropped their requirement for accreditation of hospitals to have 20% minimum autopsies. All pathologists have noted a decline by half or two-thirds of U.S. autopsies. Soon we may have so few that new diseases like legionnaires' disease cannot be effectively investigated. Recall that legionnaires' disease was blamed on cigarettes among other things, and that it was community hospital routine autopsies which permitted identification of the responsible organism.

Beside the inadequate epidemiologic matching and the defective lung cancer data from death certificates, one other event has occurred which bears upon the annual quantification of lung cancer. The rules have been changed.

In the revision of the International Causes of Death, 8th edition, called ICD-8 to ICD-9, for reporting mortality the two rubrics: Primary Lung Cancer and Lung Cancer Not Otherwise Specified (NOS) were for the first time combined. Lung cancer NOS could begin in lung or have spread to lung from many different body sites. Lung metastases are among the three most common locations for all major internal cancers. What in essence ICD-9 did was to guarantee a continued increase in the reported lung cancer mortality, conveniently disregarding that half or more of these cancers began elsewhere in the body and spread to the lungs.

Two last points about statistical epidemiology. Every textbook states them. Every active scientist knows them. Epidemiologic studies by the nature of the mathematics so far developed deal mainly with random popoulations. But smokers are self selected, as are nonsmokers. Comparisons of selected populations using mathematics valid only for random populations cannot be expected to provide valid answers.

Second, epidemiology cannot prove cause and effect. All it can demonstrate is a relationship. The nature of the relationship, causal or otherwise, has to be worked out by other methods, usually experimental.

The CTR in 1970 undertook a large scale research program to investigate whether cigarette smoking causes lung cancer in animals. Almost 14 million dollars went into the project in twelve years. To take account of heredity, inbred mice were used, and they were tested for and vaccinated against respiratory viruses. To show that these animals could develop the major types of human lung cancer, pure chemical carcinogens were introduced down their tracheas. Biochemically, these particular mice were known to metabolize these carcinogens into the biologically active forms. About 20% developed "human type" lung cancers as a result of exposure to these pure chemical carcinogens.

Thereafter, thousands of mice were exposed daily to fresh whole cigarette smoke of either low nicotine and high tar or high nicotine and high tar content up to maximum tolerance during their whole lives, up to 40 months in some cases. At one point, there were 11,000 animal manipulations per day, including sham smoking and control mice. After all these years of cigarette smoke exposure, practically zero lung cancers developed, and not one case of squamous cell carcinoma, the human cancer most often blamed on smoking. There was no question that the cigarette smoke had penetrated into their lungs, since this was worked out quantitatively.

In other experiments, mice were primed with intratracheal pure carcinogenic chemicals and then exposed to cigarette smoke during their whole lives. No increase in lung cancer occurred over the incidence found after using pure chemicals alone, and in one experiment the smoking was associated with a reduced lung cancer rate. The numbers of animals and the computerized information are so abundant that statistical analysis by modern experimental methods is still continuing. Some 40 publications have in part already appeared or are in prospect.

In summary, a massive experiment to demonstrate that cigarette smoking can cause lung cancer in animals has proved negative. One knows how important an experimental model is in cancer research from the excitement that attended the claims years ago that cancers of lung or larynx had been produced by cigarette smoke in animals. However, no model so far developed withstands an objective analysis of the pathology of the alleged cancers.

In summary, lung cancer, like many other human cancers, remains a major biological mystery. Epidemiologic studies report a statistical association between cigarette smoking and lung cancer. However, the biomedical experimentation does not support the smoking causation hypothesis.

CURRICULUM VITAE

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Editor, Pathology Annual, 1966 -; Pathology Decennials, 1975; Co-Editor,
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Statement

Professor T. D. Sterling

I am Theodor Sterling, University Research Professor at Simon Fraser University, Burnaby, British Columbia. I have also taught at the following universities: Alabama, Cincinnati, Michigan State, Princeton, and Washington University at St. Louis. I have served as an adviser to the Food and Drug Administration, the Federal Trade Commission, the National Science Foundation, the National Security Agency, the National Academy of Sciences, and the Veterans Administration. I am a Fellow of the American Association for the Advancement of Science and of the American Statistical Association. My major professional work concerns the collection and interpretation of scientific data and the design, execution and analysis of experiments and of survey studies. Much of my research has centered on learning about the effects of environmental exposures on the health of animals and men.

I wish to comment on the proposed Comprehensive Smoking Prevention Education Act of 1981. I am opposed to this proposed legislation because I believe its far-reaching "findings" do not have adequate scientific support. Further, if it becomes law, this Act could unreasonably divert attention and resources away from crucially important areas of public health research.

My comments are based on my analyses of reports of other investigators and my own research efforts in the smoking and health areas.

Smoking and Health Studies

My interest in the possible health effects of cigarette smoking goes back to the late 1950's. In a number of published articles, I have expressed my concern about many smoking and health studies, primarily because the approaches taken have oversimplified what in reality is an extremely complex problem. I have pointed out specific flaws in the design and execution of many of these studies.

The bulk of the data on smoking and health comes from studies of population groups, that is, from epidemiological studies. These data are analyzed using statistical methods to determine whether there are associations between certain factors and disease end-points. As a statistician, I am frequently astonished to see how many people, even many who should know better, treat statistical associations as proof of cause-and-effect relationships. The findings in the proposed Act seem to be a perfect example of this misuse of statistics. Scientists and nonscientists alike must heed the words of a well-known epidemiologist, who very recently said:

> Epidemiological studies, even prospective ones . . ., cannot prove cause-and-effect when the end-point, 'effect', is an outcome of a chronic non-communicable condition.[1]

In order for statistical associations to provide reliable directions for further research, the data from which they are derived must be accurate and these data must be analysed using appropriate methods, taking all possible confounding factors into account. Unfortunately, these scientific standards frequently have not been met in epidemiological studies of smoking and health.

I have evaluated in depth two of the major prospective mortality studies. The largest of these was the "million persons" study conducted by the American Cancer Society (ACS) some years ago. Although one would hope that results from such a large and expensive study would be reliable, after detailed consideration, I find that the conclusions about the possible relationship of smoking and mortality may not be valid because of the biased procedures used to select the population.[2,3]

The data from this large study population were gathered by volunteer workers, and it appears that many of the volunteers had preconceived views on smoking and disease. In other words, since the purpose of the study was to show a link between smoking and disease, the volunteers would be more likely to choose ill subjects who smoked. Indeed, some of the disease patterns of the ACS population were quite different from those of the U.S. population. For example, the ACS women had twice the percentage of breast cancer deaths and three times the percentage of lung cancer deaths, compared to U.S. females generally. Likewise, the percent of ACS males who died of lung cancer was twice that of all U.S. males. Yet the overall death rate for ACS men was about the same as that for U.S. males and that for ACS women was somewhat lower than that for U.S. women.

The ACS population was also very different from the general U.S. population with respect to age distribution, educational attainment, racial structure and place of residence. It is clear that these one million men and women were not representative of the U.S. population. Certainly, results of the ACS study cannot legitimately be projected to the general population, yet this has been and is still being done. And astonishingly, the ACS has recently announced it will soon begin a second "million persons" study, using the same procedure of subject recruitment by "dedicated" volunteers!

A smaller American mortality study, which is still widely quoted, used information obtained from U.S. veterans. I have extensively re-analyzed these data and have found that approximately twenty-five percent of the subjects had been misclassified with respect to smoking habits.[4] For example, large numbers of misclassifications were made among current smokers of more than one form of tobacco and among all categories of former smokers. These misclassification errors directly affect the number of deaths by smoking classes and number of years at risk used to determine mortality ratios. Thus, it is appropriate to ask whether conclusions based on these extensive misclassifications can possibly be valid. This question is particularly significant since the veterans' data are periodically updated and the results used as further evidence of the adverse health effects of smoking.

As everyone is aware, smoking has also been statistically linked with "excess" morbidity, that is, "excess" illness and disability. I have re-analyzed the data from the Health Interview Survey,[5] on which this claim is said to be based, and I have found that these data may not, in fact, warrant the conclusion that cigarette smoking leads to increased disease and disability.[6] Specifically, I found that female smokers by and large report fewer diseases and disabilities than female non-smokers. Also, moderate smokers, both male and female, very often report the fewest number of diseases and have the lowest prevalence rates even when compared to non-smokers. I did not use special methods to analyze the data to arrive at these conclusions -- these same observations for female smokers and moderate smokers appear clearly in the Tables in the chapter on morbidity in the 1979 Surgeon General's Report on Smoking and Health.[7]

In addition, when I examined the Health Interview Survey methods, I observed that several potential sources of bias were simply ignored. For example, much of the information was obtained from proxy responses, the method used to adjust for age distribution differences between smokers and non-smokers may have been inappropriate, and some known differences between smokers and non-smokers, such as occupation, were not considered.

The following statement, also from the morbidity chapter in the 1979 Surgeon General's Report on Smoking and Health, clearly supports my contention that studies on smoking and health have ignored possible confounding factors.

> Most large scale studies on smoking and health have tended to investigate the role of smoking independently of other behavioral variables, such as alcohol consumption and other life style factors, occupational and environmental hazards, and certain psychological factors. These variables are known to be related to health status and many are also related to smoking habits.[7]

Occupational Exposures and Smoking

For many years, I have been concerned with and have studied in depth the effects of various environmental agents on the health of man. Modern society has created a multitude of sources for toxic airborne particles and fumes. All of us carry an accumulative body burden from by-products of carbonization, from dusts, and from a variety of chemicals and substances which are released into our environment. The sources are as different as the vinyl cover of a sofa, the kitchen gas stove, the heating air duct, the automobile tail pipe, and the huge smokestacks of a steel mill. But nowhere are people exposed to such high concentrations of fumes and toxic dusts as they are in the industrial workplace.

Yet there has been comparatively little effort to collect and analyze data on occupational exposures. For example, as difficult as it may be to believe in 1982, I know of only a single cancer registry that collects appropriate detailed information on occupation and lifetime work exposure among all the many cancer registries that inquire about the smoking habits of the respiratory cancer cases in their files. It was not until 1970 that an institute (NIOSH) to study the diseases of the workplace was established. In the opinion of many, NIOSH was not funded adequately until 1976 -- and even that relatively modest funding was cut again recently. And it was not until 1980 that the American Lung Association convened a special Task Force on Occupational Health.

In contrast, there have been many, many publicly funded studies seeking to associate smoking with disease and simultaneously neglecting the environment of the smoker. Population studies such as the ACS and veterans' studies either did not obtain information on industrial exposures or, if some limited information was available, it was not subjected to critical analysis. Unless we begin to attend to the importance of the workplace and exposures to chemicals in the past, we may deprive ourselves of the ability to take effective measures to prevent the constant increases in respiratory disease rates.

Only recently have studies appeared which have addressed the question of the health experience of special groups of industrial workers. For example, asbestos workers, uranium workers and chemical workers have received attention from epidemiologists. I have observed that even many of these investigators gather data on smoking habits with much more thoroughness than they do data on type and duration of exposures. Their analyses of the observed disease patterns, in my view, place disproportionate emphasis on smoking histories in drawing conclusions.

My own investigations have justified my concern that a narrow focus on smoking has masked the effects of micro-chemical and occupational environments. I have been prompted by my results to ask: does smoking kill workers or working kill smokers?[8] Evidence in support of the latter has been detailed in a paper I presented at the American Lung Association's Occupational Health Task Force Meeting in April, 1980.[9] Here I will briefly summarize that paper.

While there are many reasons for the confusion between the possible effects of smoking and other variables, the major complication arises because the prevalence of smoking is highest among those individuals who are exposed to possible deleterious occupational factors.[10,11] For instance, 71 percent of construction painters are current smokers while only 27 percent of teachers have that habit.[11] In fact, when occupations are arranged by prevalence of smoking, 29 out of 40 occupations in which smoking is most prevalent are also those with high exposure to dusts and fumes, while in only 4 of the 40 occupations with the lowest prevalence of smoking are workers exposed to such hazards.[10]

There is a strong tendency for a higher proportion of individuals who do dirty work to smoke, compared to individuals who do clean work. Thus, in a statistical sense, the category *smoker* may be primarily an index for *worker exposed to occupational hazards*. It follows that any comparison between smokers and non-smokers implies a comparison between groups that probably differ significantly in their exposures to dusts and fumes in the workplace. In short, the increased incidence of disease, ascribed to smoking by epidemiological studies that fail to control adequately for occupation, could well be due to occupational factors. Obtaining an accurate occupational history is difficult and time consuming, particularly because important exposures may have occurred twenty or more years ago.

Studies of Industrial Workers

I turn now to a discussion of recent and long overdue studies of lung disease in occupationally exposed workers; some of these have failed to find any significant effect due to cigarette smoking, while others have found even higher disease rates among non-smokers than among smokers.

In a study of zinc and lead miners, Axelson, et al.[12] report a greater risk for lung cancer among non-smokers compared to smokers. Another study of miners' mortality by Dahlgren, reported in 1979, showed that non-smoking miners had a higher mortality from lung cancer than smoking miners.[13] Axelson emphasized that these Swedish studies deal with a lifetime follow-up of miners whereas most other mining populations have been studied by means of cohorts with a follow-up of not more than about 25-30 years or less.[14] In other words, the completeness of the follow-up leads to results with added reliability.

An inverse relationship between smoking and lung cancer was also found among workers exposed to chloromethyl methyl ether.[15] The author, W. Weiss, who is known for his militant opposition to smoking, observed: "The data suggest that continued cigarette smoking entailed a factor which partially inhibited the carcinogenic effects of chloromethyl ethers."

Pinto[16] found elevated lung cancer mortality rates in his study population of arsenic exposed workers, compared to the general population. He commented that these elevated rates were "not due to smoking" in his workers. Further, he reported that the differences between the elevated standard mortality ratios in the three groups (smokers, ex-smokers and non-smokers) were not statistically significant.

A study of chrysotile asbestos miners in Canada[17] reported that "Lung cancer deaths occurred in non-smokers, and showed a greater increase of incidence with increasing exposure than did lung cancer in smokers . . . ." This is one of the very few studies that estimated levels of asbestos dust exposure among the workers.

Some scientists have claimed that occupational exposures and cigarette smoking increase the risk of disease. In fact, Finding (6) of H.R. 4937 accepts this view. The recent studies discussed above do not support this hypothesis. In fact, Axelson and Weiss separately raised the possibility that smoking may have protective properties for some types of work.

The claim of interaction is heard most often regarding asbestos and smoking, but even here the evidence needs to be examined carefully. Although the well known study of insulation workers by Hammond and Selikoff[18,19,20] has indicated an apparently large effect on lung cancer rates due to the claimed interaction of smoking and asbestos, that "effect" has become smaller with successive reports, as increasing numbers of lung cancers occur among non-smoking workers. It is important to note that the latest report by these investigators has shown a five-fold relative risk of lung cancer mortality for smoking and non-smoking workers exposed to asbestos.

In addition, there are serious problems with the Hammond-Selikoff study. For example, there were no smoking histories available for over 6,000 of the 17,800 insulation workers. This prompted a cancer researcher to note ". . . the large fraction of subjects with unknown smoking habits makes uncertain any quantitative assessment of the joint effect of smoking and asbestos."[21] Also, I have pointed out that in these insulation workers, the proportion of deaths due to cancer of all sites was the same for both smokers and non-smokers.[8] Approximately 43% of all deaths were due to cancer in these workers. In contrast, the proportion of expected deaths from cancer (based on age-specific U.S. mortality rates for white males) was only about 18%. These figures strikingly indicate the dangers of asbestos exposure for all workers, whether they smoke or not.

Ongoing Studies

In many of these reported studies, conclusions about the possible relative effects of smoking and occupational exposures were almost an afterthought. There have been few studies designed for the express purpose of evaluating the possible interaction of smoking and occupation. In order to fill that void, a number of associates at the Sinai Hospital in Detroit and the Rhode Island Hospital of Brown University have joined me in a study seeking to untangle the possible influences of the life history of smoking and exposure to toxic dust and fumes on patients in lung, cancer, heart, dental and other hospital services. We have developed a thorough questionnaire and a practical interviewing procedure to determine whether an individual has been exposed to occupational hazards, and the extent of any such exposures.

While the analysis of all our data is not yet complete, it is nevertheless clear that the association previously claimed between smoking and respiratory cancers has been greatly magnified because other factors were not considered. The much-publicized "association" between heart disease and smoking may be similarly exaggerated. Indeed, if our preliminary findings are substantiated when data analysis is complete, the statistical association reported by others between smoking and heart disease may have to be completely re-evaluated.

Conclusions

My purpose here has been to present some results of scientific investigations that are not widely publicized, and to show that an almost exclusive focus on cigarette smoking may have seriously hindered the scientific study of chronic diseases. I believe I have clearly demonstrated that the possible effects of smoking and occupational exposures have not yet been disentangled.

As humans living in a frighteningly complex world, we grasp with relief at what appear to be simple answers. Indeed, the readiness with which the existing evidence has been accepted as demonstrating that cigarette smoking is the major antecedent for lung disease is, perhaps, a most striking example of our desire to keep our world simple and orderly.

Nature, on the other hand, is not concerned with what we believe causes disease, and the real world is not always simple. In the case of lung disease, in fact, it is highly complex. The factual knowledge about the antecedents of lung disease will remain incomplete if we continue to simplistically blame cigarette smoking and continue to ignore the possible effects of the workplace on the health of workers.

I am afraid that the bill under consideration would tend to reinforce what has been the dominant approach of health scientists -- the search for antecedents of disease in the behavior and life-styles of individuals. Attempts are constantly made to link disease to individual habits such as nutrition, exercise or the lack of it, alcohol, and, of course, smoking. For example, the new American Cancer Society study mentioned earlier is targeted on the effects of certain life-style factors on cancer.

In contrast, the idea that the day-to-day environment exerts a major influence on the health of large population groups, although suspected by some, has not been adequately considered. One needs only to recall the criticisms heaped upon Rachel Carson's Silent Spring twenty years ago to realize the truth of this statement. The fact that many people are exposed to substantial concentrations of volatile fumes and toxic dusts, particularly in the workplace, has not received sufficient attention from epidemiologists and public health officials.

In conclusion, I believe that the facts I have presented support my contention that the "findings" in the bill are based on insufficient data. This legislation would add little to present public health efforts; it would merely reinforce the state of imbalance and confusion which now exists in investigations of the health consequences of the micro-chemical environment.

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STATEMENT OF PROFESSOR YORAM J. WIND FOR SUBMISSON TO THE SUBCOMMITTEE ON HEALTH AND THE ENVIRONMENT

I am Yoram (Jerry) Wind. Since 1973 I have served as Professor of Marketing at the Wharton School of the University of Pennsylvania, and am the past editor of the Journal of Marketing. I have been on the faculty at Wharton since receiving my doctorate degree from Stanford University in 1967. My specialty is marketing research, with particular emphasis on the analysis and measurement of consumer behavior. During the past fifteen years I have served as a research consultant for various government agencies and about 100 companies and have published extensively in many areas of marketing. A resume of my educational background and professional activities, and a bibliography of my publications, are attached to this statement.

I have been asked by The Tobacco Institute to present my views, as an expert in marketing and consumer behavior, on the theoretical and factual support for certain of the provisions of H.R. 5653. My testimony will deal with the proposed findings that existing government and private programs, including the Surgeon General's warning statement, have not adequately informed the public about smoking and health issues, and the proposal to replace the current warning statement with a rotational system of seven different warning statements. My comments are based on an evaluation of a document issued in May 1981 by the Staff of the Federal Trade Commission entitled Report on the Cigarette Advertising Investigation, which I understand was submitted to Congress and was based on a rationale that appears to be similar to that underlying the labeling provisions of the present bill. I also have reviewed the consumer studies and surveys that are principally relied upon in that Report.

My conclusion can be stated in one sentence: to the extent that the labeling and advertising proposals contained in H.R. 5653 are based on the recommendations and conclusions set forth in the FTC Staff Report, those proposals are without factual or theoretical support and are unlikely to achieve the bill's objective.

I base that conclusion on two key points:

First, the level of public awareness about various smoking and health issues, as demonstrated by the studies and surveys cited in the FTC Staff Report, is extraordinarily high -- much higher than one would expect to result from normal advertising and marketing methods. The efforts of the FTC Staff to minimize the extent of public awareness on these issues are based on misinterpretation and misuse of the studies cited in the Report and a fundamental confusion between awareness and belief. The conclusion presented in the proposed bill that "present Federal, State, and private initiatives have been insufficient in conveying the health messages to the American Public" (Sec. 2(7)), is not consistent with the findings of the studies relied upon by the FTC Staff Report.

Second, there is neither theoretical nor empirical support for the proposition that the rotational warning system proposed in H.R. 5653 and recommended by the FTC Staff would have any positive impact on the level of public awareness about smoking and health issues. Replacement of the current warning statement with seven different rotational warnings thus would be totally arbitrary.

I would like to elaborate on each of these points.

I.

With respect to the existing level of public awareness, the FTC Report begins its analysis with the admission that "most people are generally aware" of the claims about smoking and health. The report cites a 1978 Gallup Opinion poll, which indicates that more than 90 percent of the public believes that smoking is hazardous to health. Similar high percentages respond affirmatively to more specific issues: over 90 percent of the public believes that heart disease has been found to be associated with smoking; almost 90 percent believes that smoking during pregnancy can affect the smoker's baby; 87 percent of adults are of the view that smoking has been found to be associated with cancer of the mouth and with chronic bronchitis; well over 80 percent either "think" or "know" that smokers are many more times as likely to develop lung cancer as nonsmokers.

These responses are remarkable. National surveys and polls consistently identify substantial segments of the American public who are unaware of major public issues and facts -- the energy crisis, the identity of the President and other public leaders -- the examples are numerous.

A measured level of 90 percent awareness can be considered "deficient" only if it is compared to a standard of perfect awareness. But it should be obvious that such a standard is both theoretically and practically impossible. The limits of human cognitive abilities and selective perception mechanisms insure that 100 percent of any group will never be aware of or in agreement about any fact or issue. That is why there is a distribution of responses in any test, particularly a test involving multiple choice questions such as the studies cited in the FTC Staff Report.

An equally important defect is the FTC Staff's fundamental misinterpretation of the results of the consumer studies on which it relies. Six major misinterpretations can be identified.

First, the Staff improperly focussed on responses to specific questions, rather than on patterns of responses. The Staff assumed throughout its discussion of public awareness that if a number of people are not aware of a specific detail about the smoking and health issue -- for example, the claim that smoking during pregnancy increases the risk of still birth and miscarriage -- those people are not aware of the general proposition that encompasses that detail -- that is, the assertion that smoking during pregnancy increases the risks of adverse effects on the baby.

This assumption is contrary to the actual results of the studies cited in the report, which show that most people are aware of all of the significant claims about smoking and health. It also violates fundamental principles about measurement of knowledge or awareness, which call for the development of an overall knowledge score or scores based on response to multiple items. Can your knowledge of a subject, let's say economics, politics or health, be assessed accurately by your response to a single question on each topic? Yet this is analogous to the FTC Staff's reliance on the response to a single question on the health effects of smoking.

The second area of misinterpretation is the Staff's assumption that anyone who responds incorrectly to multiple choice questions involving detailed statistics or medical knowledge is insufficiently aware of the fact or issue involved in the question. Respondents were asked numerous questions involving precise details, such as "Out of every hundred people who get lung cancer, how many die from it," and were provided with six alternative answers. According to the Staff, anyone who picked other than the answer that the Staff called correct -- 95 -- "did not appreciate the severity of lung cancer." In fact, however, the vast majority of respondents chose either 45, 75, or 95, indicating that they believe that lung cancer has a high mortality rate of at least 1 of every 2 of those suffering from the disease. That belief hardly supports the Staff's conclusion.

Similarly, the Staff interpreted "don't know" answers to such questions as a lack of awareness on the part of the respondent. But such an answer is equally susceptible to the interpretation that the respondent is aware of the statement presented but is unsure of the precise statistics involved. In my previous example, a person who believed that most people with lung cancer die from that disease, but who was not sure whether the correct proportion is 85, 90, 95, or 97 out of 100, might answer "don't know." The Report would erroneously have included that person in the category of those who "do not appreciate the severity of lung cancer."

Still another interpretation of the "don't know" answer is the one offered by Mark Twain in Life on the Mississippi: "I was gratified to be able to answer promptly, and I did. I said I didn't know." Recognizing this, the Interviewer's Manual of the Survey Research Center of the University of Michigan points out that the answer "I have no opinion on that" can mean merely "wait a minute, I am thinking" and advises that it is a good idea to probe all "don't know" responses. No probing was conducted in the studies relied upon by the FTC Staff.

Third, many of the so-called "incorrect" responses to the studies could have resulted from simple lack of understanding of the questions. The studies primarily relied upon in the Report on the issue of public awareness were based on telephone surveys. In such interviews, it is not reasonable to expect a high percentage of correct answers to complex questions such as those that were asked in the studies.

Let me give you an example of one such question. Imagine that I have called you out of the blue, explained who I am, and ask you a long series of questions. Even if you have remained interested and alert throughout the interview, you must respond to such questions as this one:

"How many Americans living today will eventually die from diseases related to smoking cigarettes? None, one out of two, one out of six, one out of ten, or one out of a hundred?"

The ambiguity of the phrasing -- does the word "Americans" refer to all Americans or only those who smoke? -- as well as the precise statistical answers presented turn such surveys into a guessing game rather than a test of knowledge or awareness.

Fourth, none of the studies cited in the Report included supporting data as to the reliability and validity of their findings. That is, no evidence was offered to show that the series of questions asked were a reliable measure of public awareness or knowledge about any particular issue. For example, how many respondents would provide the same answers if they were reinterviewed a few weeks later? Furthermore, no validation of the results was provided. Indeed, given the focus of the FTC Staff Report on substantive conclusions, if it were submitted to a professional publication such as the

Journal of Marketing during my editorship, or Marketing Science today, it would be rejected due to the lack of any validation procedures for the studies upon which its conclusions are based.

Fifth, neither the Staff Report nor the studies presented any norm against which to compare the survey results. In the classic text on testing, Educational Measurement, William Angoff of the Educational Testing Service states:

> "By now it has become almost axiomatic that raw scores on a test yield no meaning unless they are accompanied by relevant supplementary data that will place the score in an appropriate interpretive context."

In other words, it is impossible validly to conclude that the answers to a particular survey item represents a high, low or medium level of awareness in the absence of a standard that would show what answers are to be expected.

Sixth, and most fundamentally, the Report's conclusions, and to a large extent the studies upon which those conclusions are based, reflect a hopeless confusion of the very distinct concepts of awareness versus knowledge and belief. The Staff Report defines "unaware" as:

> ". . . those who say they do not believe true statements ; believe false statements; underestimate on a multiple choice question or answer 'don't know' or uncertain." (FTC Staff Report at p. 17 note b, emphasis added)

There is no conceptual justification for combining these five diverse responses. Belief is not awareness or knowledge; looking for exact response on a multiple choice question in search of the precise "correct" answer is not reasonable, "don't know" is both a function of the question wording and the item of concern; and uncertainty does not necessarily represent lack of awareness.

One example of this faulty definition of "awareness" is the FTC Staff's misuse of the 1980 Roper Study cited in the Report. That study asked people "how true you personally *think*" a particular statement is. The interviewer was instructed to introduce those questions to the respondents as follows: "Now I'm going to read you some statements about smoking and health, and for each one I'd like you to tell me *your beliefs* about how true the statement is." Respondents were permitted to answer only "know it's true," "think it's true," "don't know if it's true," "think it's not true," or "know it's not true." The Staff concluded that those responding in the latter three categories are "unaware" of the information conveyed by the specific statements made. It is obvious, however, that a respondent could be aware of a claim (for example, that smoking increases the risk of heart attack) yet disagree with it.

The FTC Staff consistently misused the studies in this respect to support its erroneous conclusions about awareness. Let me read another example from page 3-19 of the Report:

> "According to the Gallup Opinion Index, June, 1978, 19% of the population *do not believe* that smoking causes lung cancer . . . . Among all smokers, 28% *did not believe* smoking caused lung cancer while among heavier smokers, nearly one-third -- 31% -- *did not believe* or

> know about the link. . . . Projected nationwide, these data suggest that tens of millions of Americans, both smokers and non-smokers, do not know that cigarette smoking causes lung cancer."

The Staff's conclusion simply does not follow from the data. This fundamental type of error -- confusing consumer awareness with consumer belief -- undermines any recommendations that might be based on a premise that the public is not adequately informed about smoking and health.

II.

The FTC Staff Report -- and presumably the present bill -- proceed from the premise that the public is inadequately informed about smoking and health issues to the conclusion that a new system of warning statements is necessary to rectify this inadequacy. As I have attempted to demonstrate, the available data do not support the premise. But it is equally disturbing that the new system of warning statements recommended by the Report and by H.R. 5653 are not likely to achieve any positive impact on consumer awareness about smoking and health issues.

There is no evidence that specific warnings such as those proposed in H.R. 5653 would result in any increase in public awareness. The only study of which I am aware that relates to this issue is a study conducted for the FTC Staff by Burke Marketing Research to test the recall of different types of proposed warning statements and formats. This study has three major limitations. One, it is restricted to warning statements in cigarette advertising, not packaging. Two, the sample of respondents is not representative of the American public, and thus the projectability of the study's results is questionable. Three, the study included only two new warning statements and the current statement as a control; the two statements tested are not among those proposed by the present bill.

Given these limitations, it is clear that the study does not provide empirical support for the present proposal. But even were one to assume away the limitations and accept the results of the study as valid, they demonstrate only that consumers best recall those matters that they already had learned from the present warning statement and from the numerous other sources of information about smoking and health. Thus, for example, the study found that a specific lung cancer warning was "no more effective than the non-cancer warnings in eliciting mentions of the relationship between smoking and cancer."

Indeed, it is quite possible that the rotational warnings proposed by H.R. 5653 would have an opposite effect of what is intended by the bill's sponsors. The present Surgeon General's warning statement is embedded in the public consciousness, as well or better known than the proposition that people should wear seat belts. If instead appear statements linking smoking to specific health problems, consumers might well conclude that the Surgeon General has changed his opinion and no longer considers smoking to be generally hazardous to health. Moreover, by focussing on specific problems, the warnings become significantly less relevant to various segments of the population. Teenagers, for example, are unlikely to be concerned about emphysema or heart disease; single men and older women are not likely to pay attention to a warning that smoking may cause problems during pregnancy. Neither the FTC Staff Report nor the present bill appears to have given any consideration to these potential consequences of a rotational warning system.

III.

In conclusion, nothing in the studies cited by the FTC Staff or the rationale suggested in the bill itself supports (a) the diagnosis that the public is unaware of the claimed health hazards of smoking and (b) the prescription that the replacement of the current Surgeon General's warning with a rotational system of seven different warnings will have any positive effect on consumers' awareness about smoking and health issues. The labeling proposals simply are unsupported by facts or theories about consumer behavior. Hence, I urge this Subcommittee to reconsider the advisability of the labeling provisions of H.R. 5653.

YORAM (JERRY) WIND

Professional Resume

Dr. Yoram (Jerry) Wind is a Professor of Marketing and Director of the Wharton Center for International Management Studies at the Wharton School, University of Pennsylvania. He is an area editor of the new *Journal of Marketing Science* and the previous editor of the *Journal of Marketing*. He joined the Wharton staff in 1967 after receiving his doctoral degree from Stanford University.

He is the recipient of a number of research awards, including two Alpha Kappa Psi Foundation Awards for the best articles published in the *Journal of Marketing* in 1973 and 1976.

Dr. Wind has consulted and conducted research projects for close to 100 companies. His current clients include Pfizer Pharmaceuticals, MRCA, Pepsico, and E.F. Hutton Company. In addition, he is a frequent lecturer in faculty seminars at numerous universities and various executive programs in the U.S., Canada. Australia, Japan, Europe. South America and the Middle East.

Dr. Wind is the author of a recently published book on *Product Policy* and a co-author of several books including *Advertising Measurement and Decision Making*, *Market Segmentation*, *Organizational Buying Behavior*, and *Multi-Attribute Decisions in Marketing*. He is a regular contributor to professional marketing literature, which have included about one-hundred and fifty of his articles, monographs and chapters. His writings have encompassed a wide spectrum within marketing, with special emphasis on marketing research, product and marketing strategy, and consumer and industrial buyer behavior.

He has served on the editorial boards of all the major marketing journals and as an occasional reviewer to numerous other professional journals. He is also the consulting editor of Addison-Wesley's marketing series and the editor of the newly established Wharton Executive Library.

Dr. Wind is an active member in a number of professional associations. He is a past Chairman of the Institute of Management Science, College of Marketing and the Policy Board of the *Journal of Consumer Research*. He is a current member of the Board of Directors of the Philadelphia Chapter of the AMA and the Parlin Board of Governors (which he chaired).

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ACADEMIC EXPERIENCE

EDUCATION

Stanford University, Graduate School of Business, Ph.D. (Marketing), 9/64 - 12/66.

Stanford University, International Center for the Advancement of Management Education, Certificate in Marketing Management, 9/63 - 6/64.

The Hebrew University, Jerusalem, School of Economics and Social Sciences, M.A. (Business Administration and Political Science), 9/60 - 6/63; B.Soc.Sc. (Economics and Political Science), 9/58 - 6/61.

UNIVERSITY POSITIONS

University of Pennsylvania, The Wharton School

Professor of Marketing, July 1973 to present.
Senior Fellow of the Leonard Davis Institute, 1977 to present.
Member of the OR Graduate Group, 1979 to present.
Secondary Appointment in the Dept. of Management, 1981 to present.
Member of the Extended Faculty of the Social Systems Sciences Dept., 1981 to present.
Associate Professor of Marketing, June 1970 to June 1973.
Asst. Prof. of Marketing & International Business, January 1967 to June 1970

The University of New South Wales (Australia)

Visiting Hoover Foundation Professor, June 1977.

University of California at Berkeley, School of Business Administration

Visiting Professor, Fall 1975.

University of Tel Aviv, The Leon Recanati Graduate School of Business Admin.

Visiting Senior Lecturer, September 1968 to August 1969.

Stanford University Graduate School of Business

Research Assistant, June 1965 to September 1966.

The Hebrew University, Jerusalem

Teaching Assistant in the Departments of Policy Science and Business Administration, September 1961 to June 1963.

PERSONAL DATA

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Date of Birth: March 27, 1938

Place of Birth: Haifa, Israel

Marital Status: Married to Vardina Wind (BA in Sociology, Hebrew University, 1962; MA in Communications, University of Pennsylvania, 1974); two children, 15 and 20.

Non-Professional Affiliations:

The Institute of Contemporary Art of the University of Pennsylvania

The Friends of the Philadelphia Museum of Art

The Museum of Modern Art (New York)

Military Service:

Completed active duty in the Israeli Artillery Corps. (1956 to 1958).
Rank: Second Lieutenant

January 1982

PUBLICATIONS
OF
YORAM (JERRY) WIND

January, 1982

Professor of Marketing
The Wharton School
University of Pennsylvania

I. BOOKS AND MONOGRAPHS

*_Product Policy: Concepts, Methods & Strategies_. (Reading, MA: Addison-Wesley), 1982.

*_Marketing and Product Planning_ (in Spanish)(Mexico: Expansion). (This book is based in part on sections from _Product Policy_ which was selected by the editors of Expansion as the "Book of the Year" 1979.

Multi-Attribute Decisions in Marketing: A Measurement Approach, with Paul E. Green (and contributions by Douglas Carroll). (Hinsdale, IL: The Dryden Press, 1973).

Organizational Buying Behavior, with Frederick E. Webster, Jr. (Englewood Cliffs, NJ: Prentice-Hall, Foundations of Marketing Series, 1972). Translated to Portuguese (Editor, Atlas, Sao Paulo, Brasil, 1975). Reviewed _JMR_ August 1974.

Market Segmentation, with Ronald E. Frank and William F. Massey. (Englewood Cliffs: NJ: Prentice-Hall, International Series in Management, 1972). Reviewed _JMR_, November 1972, August 1974.

Advertising Measurement and Decision Making, with Homer Dalby and Irwin Gross. (Boston, MA: Allyn & Bacon, 1968).

Industrial Buying and Creative Marketing. Publication of the Marketing Science Institute (Boston, MA: Allyn & Bacon, 1967). Contributed two chapters; co-authored with Patrick J. Robinson three chapters; and participated in the overall organization and preparation of the book.

*_Industrial Buying Behavior: Source Loyalty in the Purchase of Industrial Components_. Unpublished Ph.D. dissertation, Stanford University, December 1966.

II. EDITED BOOKS

*_New Product Forecasting: Models and Applications_, with Vijay Mahajan and Richard Cardozo. (Lexington, MA: Lexington Books), 1981.

*_Advances in Organizational Buying Research: The Case of Acquisition of Scientific and Technical Information_, with Robert J. Thomas. Washington, DC: National Science Foundation, 1979.

*_Moving A Head with Attitude Research: Proceedings of the Seventh Attitude Research Conference_. Edited with Marshall Greenberg. (Chicago, IL: American Marketing Association.) Designed, organized, and edited this book of eight invited position papers and eighteen application papers, 1977.

Behavioral Models of Market Analysis: Foundations for Marketing Action. Edited with Francesco Nicosia. (Hinsdale, IL: The Dryden Press). Co-planned, edited and organized the volume of original papers, 1977.

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*Single or Senior Author

III. ARTICLES, CONTRIBUTED CHAPTERS, PROCEEDINGS, PAPERS AND WORKING PAPERS

The papers in this section are classified into five areas:

- research on industrial buying behavior
- research on consumer behavior
- marketing and product management and strategy
- marketing research
- international marketing

A. Research on Industrial Buying Behavior

1. *"The Determinants of Vendor Selection: An Evaluation Function Approach," with Paul E. Green and Patrick J. Robinson, Journal of Purchasing, 4 (August 1968) 29-41.

2. *"Applying the Behavioral Theory of the Firm to Industrial Buying Decisions," The Economic and Business Bulletin, 20 (Spring 1968), 22-28.

3. *"Integrating Attitude Measures in a Study of Industrial Buying Behavior," in Lee Adler and Irving Crespi, eds., Attitude Research on the Rocks. Chicago, IL: American Marketing Association, 1968, 58-77.

4. *"Mathematical Analysis of Perception and Preferences for Industrial Marketing," in K. Cox and B.M. Enis, eds., A New Measure of Responsibility for Marketing. Proceedings of the International Marketing Conference of the American Marketing Association, June 1968, 284-294.

5. "Generalized Simulation of the Industrial Buying Process," with Patrick J. Robinson, Marketing Science Insitute, Working Paper, P-46-2, June 1968.

6. *"Simulating the Industrial Buying Process," with Patrick J. Robinson, in R.L. King ed., Marketing and the New Science of Planning. Proceedings of the American Marketing Association, August 1968 Conference, 441-448.

7. *"Computer Simulation: Marketing Management Tool," with Patrick J. Robinson, Computer Operations, 3 (January-February 1969), 42-47.

8. *"Industrial Source Loyalty," Journal of Marketing Research, 7 (November 1970), 450-457.

9. *"A Reward Balance Model of Buying Behavior in Organizations," in George Fisk, ed., New Essays in Marketing Theory. Boston, MA: Allyn & Bacon, 1971, 206-217.

10. "Industrial Marketing Research," with Richard N. Cardozo. Paper presented at the AMA Workshop on Industrial Buying Behavior, Berkeley, CA, April 1971.

11. "A General Model for Understanding Organizational Buying Behavior," with Frederick E. Webster, Journal of Marketing, 36 (April 1972), 12-19.

12. *"On the Study of Industrial Buying Behavior: Current Practices and Future Trends," with Frederick E. Webster, Industrial Marketing Management, 1 (July 1972), 411-416

13. *"Industrial Buying As Organizational Behavior: A Guideline for Research Strategy," with Frederick E. Webster, Journal of Purchasing, 8 (August 1972), 5-16.

14. *"Segmenting Media Buyers," with Steve Silver, Journal of Advertising Research, 8 (December 1973), 33-38.

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| Article

15. *"The Industrial Customer," with Elmer Lotshaw, in Stuart H. Britt, ed., Marketing Handbook. The Dartnell Corp. 1973, 781-792.

16. *"Recent Approaches to the Study of Organizational Buying Behavior," in T.V. Greer, ed., Increasing Marketing Productivity. Proceedings of the American Marketing Association, April 1973 Conference, 203-206.

17. *"Industrial Marketing Segmentation," with Richard N. Cardozo, Industrial Marketing Management, 3 (March 1974), 153-165. (Also appeared in the French Encyclopedie Du Marketing, 1978).

18. "Marketing Research in the Design of STI Systems: A Case Study," with John F.Grashof, in R. Mason and J. Kreps,Jr.,eds. Information Services: Economics, Management and Technolo Boulder, CO: Westview Press, 1981, 73-84.

19. "Emerging Models of Organizational Buying Processes," with Francesco M. Nicosia, Industrial Marketing Management, 1977, 6, 353-369, and in Nicosia and Wind, eds., Behavioral Models of Market Analysis: Foundations for Marketing Action. Hinsdale, IL: The Dryden Press, 1977, 96-120.

20. *"Information Requirements on Buying and Usage of STI Services," in Current Research on Scientific and Technical Information Transfer. New York: J. Norton Publishers, 1977 - A Micropapers Edition.

21. *"Market Based Guidelines for Design of Industrial Products", with John F. Grashof and Joel D. Goldhar, Journal of Marketing, 14, July 1978, 27-37.

22. *"The Boundaries of Buying Decision Centers," Journal of Purchasing and Materials Management, 14, Summer 1978, 23-29.

23. *"Organizational Buying Center: A Research Agenda," in Thomas V. Bonoma and Gerald Zaltman, eds., Organizational Buying Behavior. Chicago, IL: American Marketing Association, 1978, 67-76.

24. *"Organizational Buying Behavior," Annual Review of Marketing, 1, 1978, 160-193.

25. *"Problems and Prospects in the Segmentation of the STI Market," with Robert Thomas in William R. King and Gerald Zaltman, eds., Marketing Scientific and Technical Information, 1979, 67-76.

26. "Organizational Psychographics and Innovativeness," with Thomas S. Robertson, Journal of Consumer Research, June, 1980, 24-31.

27. *"Industrial Market Segmentation Under Conditions of Intra-Organizational Heterogeneity," in Yoram Wind and Robert Thomas, eds. Advances in Organizational Buying Behavior, Washington, D.C.: NSF, 1979.

28. *"Conceptual and Methodological Issues in Organizational Buying Behavior," with Robert J. Thomas, European Journal of Marketing, Vol. 14, 1980, 239-263.

29. *"The Linking Pin Role of Organizational Buying Centers," with Thomas Robertson, Special Issue of Journal of Business Research, 1981.

30. "Organizational Cosmopolitaness and Innovativeness," with Thomas Robertson, The Wharton School Working Paper, July 1980.

31. "Toward Empirical Generalizations on Industrial Market Segmentation," with Robert J. Thomas in R. Spekman and D. Wilson, eds., Industrial Marketing: A view to the Future. Chicago: AMA 1981.

B. Research on Consumer Behavior

1. *"Inter-Product Household Loyalty to Brands," with Ronald E. Frank, Journal of Marketing Research, 6 (November 1969), 434-435.

2. *"Incongruency of Socioeconomic Variables and Buying Behavior," in P.R. McDonald, ed., Marketing Involvement in Society and the Economy. Proceedings of the American Marketing Association, August 1969 Conference, 362-367.

3. *"Models of Customer Behavior," Organization and Administration (Hebrew), 16 (May 1970), 3-13.

4. *"The Application of Multidimensional Scaling in Segmentation Research." Paper presented at the first annual meeting of the Association for Consumer Research, Amherst, MA, August 1970.

5. "Prediction Experiments Utilizing Perceptual and Preference Judgments," with Paul E. Green. Paper presented at the Second Annual Meeting of the American Institute for Decision Sciences, Dallas, TX, November 1970.

6. *"Life Style Analysis: A New Approach," in Fred C. Allvin, ed., Marketing in Motion. Proceedings of the 54th International Marketing Congress of the American Marketing Association, April 1971, 303-305.

7. "Intentions to Buy as Predictors of Buying Behavior," with Susan P. Douglas, in D. Gardner, ed., Proceedings of the Second Annual Conference of the Association for Consumer Research, Maryland, 1971, 331-343.

8. "Benefit Bundle Analysis," with Paul E. Green and Arun K. Jain, Journal of Advertising Research, 12 (April 1972), 31-36.

9. "Subjective Evaluation Models and Conjoint Measurement," with Paul E. Green and Frank Carmone, Behavioral Science, 18 (May 1972), 288-299.

10. "A Note on the Measurement of Social-Psychological Belief Systems," with Paul E. Green and Arun K. Jain, Journal of Marketing Research, 9 (May 1972), 204-208.

11. "Experiments in the Multidimensional Psychophysics of Taste and Semantic Descriptions," with Paul E. Green. The paper was awarded first prize in the 1972 Research Design Competition of the American Psychological Association, Division 23, July 1972.

12. "Consumer Menu Preferences: An Application of Additive Conjoint Measurement," with Paul E. Green and Arun K. Jain, in M. Venkatesan, ed., Proceedings of Third Annual Conference of the Association for Consumer Research. Chicago, IL, 1972, 304-313.

13. "Preference Measurement of Item Collections," with Paul E. Green and Arun K. Jain, Journal of Marketing Research, 9 (November 1972), 371-377.

14. *"On the Teaching of Consumer Behavior: A Managerial Approach." Paper presented at the AMA August 1972 Conference, Houston, TX.

15. "Analyzing Free Response Data in Marketing Research," with Paul E. Green and Arun K. Jain, Journal of Marketing Research, 10 (February 1973), 45-52.

16. "Consumer Evaluation of Discount Cards: A Conceptual Model and Experimental Test," with Paul E. Green and Frank Carmone, Journal of Retailing, 49 (Spring 1973), 10-22.

17. "Benefit Congruence Analysis," with Paul E. Green and Arun K. Jain. Paper presented ~~at the 1974 Southeast~~ AIDS Conference.

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18. *"Some Conceptual Measurement and Analytical Problems in Life Style Research," with Paul E. Green, in William Wells, eds., Life Style and Psychographics. Chicago, IL: American Marketing Association, 1974, 97-126.

19. "Brand-Features Congruence Mapping," with Paul E. Green and Henry J. Claycamp, Journal of Marketing Research, 12 (August 1975), 306-313.

20. "On the Usage of 'Modified' Personality Trait Measures in Consumer Research," with Kathryn Villani, Journal of Consumer Research, 2 (December 1975), 223-228.

21. "Recent Approaches to the Modeling of Individuals' Subjective Evaluations," with Paul E. Green, in Philip Levine, ed., Attitude Research Bridges the Atlantic. Chicago, IL: American Marketing Association, 1975, 123-133.

22. "Alternative Approaches to Data Collection for Attitude Measurement," with Terry C. Gleason. Paper presented at the American Psychological Association Convention, September, 1975.

23. *"Multi-person Influence and Usage Occasions as Determinants of Brand Choice." Paper presented at the August 1975 AMA Conference, Rochester, NY.

24. *"Preference of Relevant Others and Individual Choice Models," Journal of Consumer Research, 3 (June 1976), 50-57.

25. *"On the Relationship Between Knowledge and Preference," with Michael DeVita, in Kenneth L. Bernhardt, ed., Proceedings of the AMA 1976 Conference, 153-157.

26. *"A 1975 Retrospective View of Bourne's 1957 'Group Influence in Marketing,'" in Louis E. Boone, ed., Classics in Consumer Behavior. Tulsa, Oklahoma: The Petroleum Publishing Company, 1977, 223-235.

27. *"Brand Loyalty and Vulnerability," in Arch G. Woodside, Jagdish N. Sheth, and Peter D. Bennett, eds., Consumer and Industrial Buying Behavior. New York: Elsevier, North Holland, Inc., 1977, 313-319.

28. *"Reflections on Creativity and Relevance of Consumer Research," in B.A. Greenberg and D.A. Bellenger, eds., Contemporary Marketing Thought. Proceedings of the 1977 AMA Conference, 33-38.

29. *"Brand Choice," in Robert Ferber, ed., Selected Aspects of Consumer Behavior. Washington, DC: U.S. Government Printing Office, 1977, 235-238.

30. "Sociology of Consumption and Tradeoff Models in Consumer Public Policy," with Francesco M. Nicosia, in Research for Consumer Policy, W.M. Denney and R. T. Lund, eds., Cambridge, MA: Center for Policy Alternatives, MIT, 1978, 141-184.

31. *"On the Interface Between Organizational and Consumer Buying Behavior," in H. Keith Hunt, ed., Advances in Consumer Research, 1977, 657-662.

32. "Examining Family Role and Authority Patterns: Two Methodological Issues," with Susan P. Douglas, Journal of Marriage and the Family, February 1978, 35-47.

33. *"A Comparison of Three Brand Evaluation Procedures," with Joseph Denny and Arthur Cunningham, Public Opinion Quarterly, 43, Summer 1979, 261-270.

34. "A Time-Money Tradeoff (TMT) Model of Consumer Behavior." with John Deighton and Francesco Nicosia (Based in part on a paper presented by me at the August 1978 APA Conference, Toronto) Wharton School Working Paper, November 1981.

35. *"On the Measurement of Purchase Data: Surveys Vs. Purchase Diaries," with David Learner, Journal of Marketing Research, XVI. February 1979, 39-41.

36. "Physical and Social Psychological Anxiety As Correlates of Purchase Behavior," with Cynthia Fraser, The Wharton School Working Paper, Revised 1980.

37. *"Comparative Consumer Research: The Next Frontier?" with Susan P. Douglas, The European Journal of Marketing, Forthcoming 1981.

38. *"A Note on the Relationship Between Perceived Problem Severity and Frequency of Occurrence," with Peter Shube, Journal of the Professional Marketing Research Society, Fall 1981.

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C. Marketing and Product Management and Strategy

1. "Innovation in Marketing: Organizational Behavior Perspective, with Kenneth E. Knight, California Management Review, 11 (Fall 1968), 67-78.

2. *"Management and Change," with Bent Stidsen and Kenneth E. Knight, Manpower and Applied Psychology, 2 (Winter 1968), 38-46.

3. "The Marketing Concept Revisited," with Thomas Schutte, The Wharton School Working Paper, January 1968.

4. *"Innovation As Marketing Orientation," Business Economics (Hebrew), 38, December, 1969, 5-11.

5. *"A Marketing Approach to the Salesman Function," Organization and Administration (Hebrew), 15 (September 1969), 26-33.

6. *"The Stepchildren of Marketing: Organizational and International Customers," The Wharton Quarterly, 7 (Fall 1972), 43-46.

7. *"A Note on the Operationalization of the Product Life Cycle Concept," The Wharton School Working Paper, July 1974.

8. *"Product Portfolio: A New Approach to the Product Mix Decision," in Ronald C. Curhan, ed., Combined Proceedings AMA, August 1974, 460-464.

9. *"Planning Product Line Strategy: A Matrix Approach," with Henry J. Claycamp, Journal of Marketing, 40 (January 1976), 2-9. (The Alpha Kappa Psi Foundation Award for the best article published in the Journal of Marketing in 1976.)

10. "Consumer Attitudes as Guidelines for the Evaluation of a New Distribution System," with Frank J. Cacchione, Jr., and Daniel Gross in Y. Wind and M. Greenberg, Moving A Head with Attitude. Chicago, IL: AMA, 1977, 139-143.

11. *"On the Use of Attitude Research in Product Policy," with Tyzoon Tyebjee, in Y. Wind and M. Greenberg, eds., Moving a Head With Attitude Research. Chicago, IL: AMA, 1977, 147-156.

12. *"The Perception of the Firm's Competitive Position," in Nicosia and Wind, eds., Behavioral Models of Market Analysis: Foundations for Marketing Action. Hinsdale, IL: The Dryden Press, 1977, 163-181.

13. *"Toward A Change in the Focus of Marketing Analysis: From a Single Brand to An Assortment of Brands," Journal of Marketing, October 1977, 12-13.

14. *"On the Multi-Dimensionality of Market Share," with Peter T. FitzRoy, Wharton School Working Paper, April 1979.

15. *"Product-Marketing Planning Models: Concepts, Techniques, and Needed Development," in Allan D. Shocker, ed., Analytic Approaches to Product and Marketing Planning. Cambridge, MA: MSI, 1979, 39-66.

16. *"A Research Program for A Marketing Guided Approach to Mergers and Acquisitions, in N. Beckwith et al., 1979 Educator's Conference Proceedings. Chicago, IL: AMA, 1979, 24-28.

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17. *"An Analytical Hierarchy Process for the Allocation of Resources Within A Target Product/Market/Distribution Portfolio." with Daniel Gross, in D.B. Montgomery and D.R. Wittink, eds., Proceedings of the ORSA/TIMS Conference on Marketing Measurement and Analysis, Stanford, CA, March 1979.

18. *"Implications of Increased Government Regulation for the Quality and Relevance of Marketing Research," in M.P. Mokwa and S.E. Permut, eds., Government Marketing: Theory and Practice, 266-289, New York: Praeger Publishers, 1981.

19. *"Marketing Oriented Strategic Planning Models," in R.L. Schultz and A.A. Zoltners, eds., Marketing Decision Models, New York: Elsevier, North Holland, 1981.

20. *"Going to Market: New Twists for Some Old Tricks," The Wharton Magazine, 4, Spring, 1980, 34-39. Based on "Product Positioning and Market Segmentation: Marketing and Corporate Perspectives," Wharton School Working Paper, 1979.

21. "Applying a Risk/Return Approach to Portfolio Analysis and Strategy," with Richard Cardozo, Wharton School Working Paper, Revised 1981.

22. *"Marketing and the Other Business Functions," in J. Sheth, ed., Research in Marketing, Vol. V, 1981, 237-[illegible].

23. *"Designing Product and Business Portfolios," with Vijay Mahajan, Harvard Business Review, Vol. 59, No. 1, Jan.-Feb. 1981, 155-163. Based on a paper, "Measurement Issues in Portfolio Analysis," presented at the Second Market Measurement and Analysis Conference, University of Texas, Austin, March 1980.

24. *"Market Share: Concepts, Findings, and Directions for Future Research," with Vijay Mahajan, Review of Marketing, B. Enis and K. Roering, eds., Chicago: AMA, 31-42, 1981.

25. *"Marketing and Corporate Strategy: Problems and Perspectives," The 13th Annual Albert Wesley Frey Lecture. Graduate School of Business, University of Pittsburgh, 1981. A revised version is to appear in the Wharton Magazine, 1982.

26. "Marketing Strategy," with Thomas Robertson, in Kenneth J. Albert, ed., Handbook of Business Strategy, New York: McGraw Hill, Forthcoming 1982.

27. *"Empirical Comparison of Standardized Portfolio Models," with Vijay Mahajan and Donald Swire. Paper presented at the 1981 Analytic Approaches to Product and Marketing Planning Conference, October 1981.

28. "Analytic Hierarchy Process for Generation and Evaluation of Marketing Mix Strategies." with Elizabeth Dunn, Wharton School Working Paper, September 1981.

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c. Marketing Research

1. "The Use of Interactive Models as Framework for Research," with Charles S. Goodman, Management Science Center, University of Pennsylvania, May 1968.

2. *"Some Applications of Mathematical Analysis of Perception and Preference in Advertising," with Patrick J. Robinson, Marketing Science Institute Special Report, No. P-43-1, May 1968.

3. *"Marketing Performance: A Conceptual Framework," with Susan P. Douglas, Marketing Science Institute Working Paper, P-31-1, Revised June 1968.

4. *"Organizing a Diffusion System for Managerial Information in Israel: A Proposal," Organization and Administration (Hebrew), 13 (September 1969).

5. *"Experimentation As A Tool for the Retailer," with Susan P. Douglas and Aaron Ascoli: Journal of the Marketing Research Society, 13 (July 1971), 138-149.

6. *"On the Measurement of Quality of Life," with Francesco M. Nicosia. Paper presented at the TIMS Conference, July 1972.

7. *"Product Positioning: An Application of Multidimensional Scaling," with Patrick J. Robinson, in R.I. Haley, ed., Attitude Research in Transition. Chicago: AMA, 1972, 155-175.

8. *"A New Procedure for Concept Evaluation," Journal of Marketing, 37 (October 1973), 2-11. (The Alpha Kappa Psi Foundation Award for the best article published in the Journal of Marketing in 1973.)

9. *"Higher Order Factor Analysis in the Classification of Psychographic Variables," with Paul E. Green and Arun K. Jain, Journal of the Marketing Research Society, 15 (October 1973), 224-232.

10. *"Multivariate Analysis of Variance in Research on the Effectiveness of TV Commercials," with Joseph Denny, Journal of Marketing Research, 11 (May 1974), 136-142.

11. *"A Note on the Classification and Evaluation of New Product Forecasting Models." Paper presented at the April 1974 AMA Conference.

12. *"Is 'Concept Testing: An Appropriate Approach' Really Appropriate?" Wharton School Working Paper, May 1975.

13. *"Concept Testing As Input to Strategic Marketing Simulations," with Stuart Jolly and Arthur O'Connor, in E. Mazze, ed., Proceedings of the 58th International AMA Conference, April 1975, 120-124.

14. "New Way to Measure Consumers' Judgments," with Paul E. Green, Harvard Business Review 53 (July-August 1975), 107-117.

15. *"Analytical Approach to Marketing Decisions in Health Care Organizations," with Lawrence K. Spitz. Special Health Care issue of Operations Research, 24 (September-October 1976), 973-990.

16. *"Determination of the Size and Allocation of Marketing Research Budgets," with Daniel Green, in Subhash C. Jain, ed., Proceedings of the August 1978 AMA Conference, 57-61.

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17. *"New Developments in Conjoint Analysis." Paper presented at the 25th Annual Midwest Conference of the *American Statistical Association* on What's New in Statistical Techniques for Marketing Research. March 1978.

18. *"Issues and Advances in Segmentation Research," *Journal of Marketing Research*, 15, August 1978, 317-337.

19. *"Marketing Research and Management: A Retrospective View of the Contributions of Paul E. Green," in A.R. Andreasen and David M. Gardner, eds., *Diffusing Marketing Theory and Research*. Proceedings of the Tenth Pual D. Coverse Award Symposium.

20. *"A Note on the Selection of Attirubtes for Conjoint Analysis," with John G. Myers, Working Paper, Revised January 1979.

21. "Product Testing: Current Methods and Needed Developments," with Randall Batsell. *Journal of the Market Research Society*, 22, April, 1980, 115-139.

22. *"Marketing Applications of the Analytic Hierarchy Process," with Thomas L. Saaty, *Management Science*, 26, July 1980, 641-658.

23. "Overlapping Clustering: A New Methodology for Product Positioning" with Phipps Arabie, Wayne DeSarbo and J. Douglas Carroll, *Journal of Marketing Research*, 310-317, XVIII, August 1981.

24. *"A Reexamination of New Product Forecasting Models," with Vijay Mahajan, in *Proceedings of the August 1981 AMA Educator's Conference*, 358-363.

25. "Stochastic Dominance Rules for Product Portfolio Analysis," with Vijay Mahajan and John W. Bradford. Marketing Planning Models, Special Issue of *TIMS Studies in the Management Sciences*, Forthcoming.

26. *"Industrial Product Diffusion by Segments," with Thomas Robertson and Cynthia Fraser, *Industrial Marketing Management*, 11, No. 1, 1982.

27. "A Model for the Analysis of Asymmetric Data in Marketing Research," with Richard Harshman and Paul E. Green, *Marketing Science*, Forthcoming 1982.

28. "TOBIT Analysis: An Alternative to Discriminant Analysis and Ordinary Least Square Regression Given Limited Dependent Variables," with Cynthia Fraser, Working Paper, April 1981.

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E. International Marketing

1. *"The Role of Marketing in Israel," Journal of Marketing, 31 (April 1967), 33-37.

2. *"Information Requirements for International Business Decisions," Background note prepared for and used in the introductory International Business course at The Wharton School, University of Pennsylvania, June 1967.

3. *"Cross Cultural Analysis of Consumer Behavior," in R. Moyer, ed., Changing Marketing Systems. Proceedings of the American Marketing Association, December 1967 Conference, 183-185. (Reprinted in the Wharton Quarterly, 2 (Winter-Spring 1968).

4. *"Marketing in Israel," in Harper W. Boyd, Jr., ed., Comparative Marketing. A collection of papers assembled by the International Center for the Advancement Education, Stanford University, Stanford, CA 1968.

5. *"Perceptual and Preference Mapping of Countries: An Application of Multidimensional Scaling," with Patrick J. Robinson. Paper presented at the Annual Meeting of the Association for Education in International Business, December 1970.

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16. *"International Portfolio Analysis and Strategy: The Challenge of the 80's", with Susan P. Douglas, Journal of International Business Studies, Fall 1981, 69-82

17 *"Marketing Perspective for International Diversification and Risk Management" with Vijay Mahajan, in Richard Herring, ed., Managing International Risk, Forthcoming 198

IV. EDITORIALS

Journal of Consumer Research

June 1977:	"New Directions for *JCR*"

Marketing News

July 1978:	"New Directions for *JM*"

Journal of Marketing

1979:	January:	"The *Journal of Marketing* At A crossroad"
	Spring:	"Repositioning the *Journal*"
	Summer:	"Bridging the Gap Between Practitioners and Academicians"
	Fall:	"On the Status of Marketing Theory"
1980:	Winter:	"Markting in the 80's"
	Spring:	"Strategic Planning and Marketing: Time for A Constructive Partnership" with George Day
	Summer:	"International Marketing: The Neglect Continuous" with John Farley
	Fall:	"Industrial Marketing: The Sleeping Giant" with Frederick Webster
1981:	Winter:	"Journals and the Development of A Discipline"
	Spring:	"Research and Management"
	Summer:	"A Positive Perspective on Marketing"
	Fall:	"Reflections"

V. PUBLISHED ABSTRACTS OF PAPERS DELIVERED IN PROFESSIONAL MEETINGS

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*"Diagnosing Consumer Behavior: A Quantitative Approach," in D. Rothwell, ed., *Proceedings of the 30th Annual AAPOR Conference*, May 1975 and in *Public Opinion Quarterly*, 39 (Fall 1975), 415.

*"Segmentation and Positioning of Health Insurance Services Under Conditions of Heterogeneous Health Insurance Portfolios," in TIMS/ORSA Bulletin for the S.F. Joint Meeting, May 1977, 244.

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*"Standardized Portfolio Models: An Empirical Comparison of Business Classifications," with Vijay Mahajan and Donald J. Swire in Allan D. Shocker, and R. Srivastava, eds., *Proceedings of the 1981 Analytical Approaches to Product and Marketing Planning Conference*.

VI. CASE STUDIES

During the academic year 1962/63 I wrote a number of marketing cases at the Hebrew University, Jerusalem (Israel). One of these cases, The Ozi Ballpoint Pens III, was published in Harper W. Boyd, Jr., et al., eds., *Marketing Management: Cases from the Emerging Countries*. Reading, MA: Addison-Wesley Publishing Company, 1966.

During the academic year 1968/69 several marketing cases were written under my supervision at the Leon Recanati Graduate School of Business Administration, Tel Aviv University.

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FOR USE AT 10 A.M. TUESDAY, MARCH 16, 1982

WASHINGTON, D.C. -- The cigarette industry's chief spokesman told a Senate committee today that he is opposed to amendments to the Federal Cigarette Labeling Act because people already "are in a position to make a free and informed choice of whether or not to smoke."

Edward A. Horrigan, Jr., chairman of R.J. Reynolds Tobacco Co. and of the executive committee of The Tobacco Institute, asked the Senate Labor and Human Resources Committee, "Why, then, should it be necessary to substitute a questionable, unproven system for the present law?"

Horrigan led a panel of witnesses testifying on a bill to substitute several specific warnings for the present Surgeon General's health warning which has appeared on cigarette packs and in cigarette advertising for more than ten years.

He said over 90 percent of Americans are aware of the claim that smoking is harmful. From the viewpoint of proponents of the bill, he added, the proposed rotational warning system may be "counterproductive."

-more-

"There is also strong evidence from the standpoint of behavioral science, based upon the evidence available in the studies cited by the Federal Trade Commission staff in support of rotated warnings, that such a system might, in fact, erode awareness," Horrigan testified.

He said the Swedish government began five years ago trying a rotating warning system which is a model for the pending bill, and noted a Swedish report that cigarette consumption has continued to rise in that country.

Horrigan challenged the scientific basis for the proposed legislation.

"After three decades of investigation," he told the committee, "no causal link between smoking and disease has been established. All of the statements presented as established fact in this bill have been challenged by research findings of many eminent scientists."

He added that "the public has been made aware of the so-called health hazards of smoking and that people are in a position to make a free and informed choice of whether or not to smoke."

Horrigan also took exception to a provision of the bill to require disclosure of tar, nicotine and carbon monoxide yields from cigarette smoke on packages and in advertising.

He pointed out that tar and nicotine levels have been reported to consumers in every cigarette advertisement since 1970.

"With respect to carbon monoxide," he declared, "the written submissions of scientific experts provided to you will show that carbon monoxide exposure through cigarette smoking is not a significant health issue."

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STATEMENT
OF
ROBERT CASAD HOCKETT

I am Research Director of The Council for Tobacco Research -- U.S.A., Inc. I have been employed by the council and its predecessor, the Tobacco Industry Research Committee, since 1954, first as Associate Scientific Director, then Acting Scientific Director, and in my present capacity. My Curriculum Vitae is attached.

In my opinion, the proposed amendment to the Federal Cigarette Labeling and Advertising Act will cause justifiable consternation among many earnest and able investigators of cancer, heart diseases and emphysema at the experimental and clinical levels. These scientists labor in the hope of clarifying the etiology and pathogenic steps and stages in the development of these disorders with a view to preventing or delaying their clinical appearance. Contrary to the "findings" in the bill, however, the cause or causes of these diseases have not been scientifically established, and much work remains to be done.

Statistical Association

The claims against smoking are based largely on epidemiologic studies. Positive statistical association, however, -- as epidemiologists are aware -- does not mean causation. Accordingly, these studies do not support the "findings" in the proposed amendment.

Students of logic have long recognized that a negative statistical association between an event and a putative cause may be conclusive. Thus when it was suggested that some hair dyes might produce cancers in women, a study of 5,000 beauticians who had been exposed to such dyes for twenty years showed that these persons had not experienced any excess of cancer. This negative correlation appears quite conclusive with respect to dyes that have long been in use. Similarly a concern regarding the SV-40 virus, which causes bladder cancer in monkeys and was found to be present in some polio vaccines, was considered resolved when no higher cancer rate was found in the vaccinated humans than in the unvaccinated ones.

On the other hand, a positive statistical association between an exposure and a disease condition is well known to be only evidence of some sort of a relationship, the nature a of which remains to be discovered. For example, there is a statistical association of severe overweight in women with higher uterine- and ovarian cancer rates and in men, of higher colon-rectum and prostatic cancer. In the same category is the positive correlation between early marriage (or sexual activity) of women and eventual cancer of the cervix. Such positive associations only indicate a need for other kinds of investigation in order to elucidate the nature of the relationship.

Still more striking is the reported epidemiological finding that men and women who average seven hours of sleep per night have lower death rates from coronary heart disease than those getting either more or less sleep. Those getting less than five hours had very high death rates and those sleeping ten hours or more per night had higher than average rates.

I have previously pointed out that merely changing the hours in bed of the short-term or long-term sleepers to seven per night, would be unlikely to change this picture radically. More likely the amount of "useful sleep" that an individual can achieve is a reflection of a psychophysiological diathesis that may be the basis of the difference and which may or may not be alterable. Studies show that many persons are able to achieve "useful and effective sleep" in a relatively short period. Others may have difficulty in getting to sleep or sleeping restfully on account of psychological tensions or emotional problems. Whether such persons can be treated to reduce such tensions is a question many investigators are attempting to attack. Surveys have shown that many smokers testify to the feeling that smoking cigarettes helps to arouse them when they are drowsy but to relax them when they are tense. Confirmation of these contradictory impressions by objective pharmacological methods has been difficult, but studies of electro-encephalograms have claimed correlations between certain types of brain waves and psychological characteristics of the subject. Some subjects can learn to produce various types of brain waves (electro-encephalograms) at will. Others have difficulty in producing the types reputed to reflect inner repose. Smoking has been claimed to promote this kind of tranquility but the evidence so far is widely regarded as "soft". The discovery of centrally active small peptides (endorphins, encephalins and related substances) may eventually provide an answer to these questions.

Lung Cancer Diagnosis

In 1912, a book by Adler called attention to the occurrence of primary carcinoma in the lung and apparently created the impression that this disease was a new development whose cause must be sought. By coincidence this book appeared near the time when development of the blended cigarette in the United States was stimulating a vast expansion of cigarette use in this country, which was further stimulated by the social conditions brought about by World War I. Thus, a basis was laid for the claim promulgated a few years later that an "epidemic" of lung carcinoma had been engendered by cigarette smoking.

Meanwhile, however, the late Dr. Milton B. Rosenblatt had made a very intensive and extensive study of lung cancer as described in the medical literature, mostly European, of the nineteenth century where the art and science of pathology was developed and practiced relatively early. The application of post-mortem examinations for cause of death was made quite routinely in some central European hospitals long before it became prevalent in other areas. Carcinoma of the lung was recognized, post mortem, in a great many cases where it had been missed altogether in clincial pre-mortem diagnoses. In fact, it was missed clinically as often as 90-95% of the time. In hospitals where post mortem histological diagnosis was practiced regularly, carcinoma of the lung constituted a proportion of total male cancers similar to or greater than that reported today. Yet in that era cigarettes were practically unknown.

In retrospect, it is not strange that lung cancer was missed in clinical diagnosis. Tuberculosis was common as a source of bleeding from the lung and death from pneumonia generally occurred in the lung carcinoma patient so that cancer was not suspected.

As better clinical diagnostic methods came into use, such as radiography, bronchoscopy, exfoliative cytology, exploratory thoracotomy and others, the gap between clinical diagnoses and post-mortem discovery of lung cancer was closed gradually over several decades. The introduction of antibiotics eventually played a role also since a pneumonia might be resolved, exposing a carcinoma to discovery.

In the United States, this European work was apparently little known. The dogma was that all lung cancer must be metastatic so that the primary lesion must be found if the origin of the neoplasm was to be known. Moreover, post mortem examination was much less frequently made than in Europe.

Although histological examination of cancer tissue by an experienced pathologist still provides the most reliable diagnosis available, the development of clinical methods that are quicker and easier apparently discouraged the application of painstaking post-mortem study in this country, and has promoted the general use of less reliable clinical methods.

The lung is a frequent target for metastases from other organs. Dr. Shields Warren reported that about one-third of all cancers arising in other regions of the body eventually metastasize to the lung, often at a early stage. Dr. Rosenblatt thought it very unlikely that such metastatic cancer present in the lung but not originated there was "caused" by tobacco smoke. He felt it logical to exclude metastatic cancer in the lung from any statistical study of correlation with smoking. But the distinction between primary and metastatic cancers in the lung is not always easy and a distinction is no longer required on death certificates or attempted in statistical records. While the distinction appears to be very important for the study of causative factors and etiology the necessary data are not easily available.

Nevertheless in a careful review of contemporary death certificates and hospital records in a few individual hospitals, Rosenblatt found considerable clinical over-diagnosis of lung carcinoma in patients who were known to be cigarette smokers. Similar findings have been made by Feinstein.

Animal Experiments

When the first large-scale epidemiological studies of the association between cigarette smoking and lung carcinoma were described publicly in the mid-fifties, a number of investigators undertook to expose many different species of animals, including mice, rats, hamsters and hens, to cigarette smoke inhalation. None of these early inhalation studies produced lung carcinomas. The 1958 Annual Report of the British Empire Cancer Campaign referred to these experiments as adding up to a "striking negative result."

The first wave of smoke inhalation studies was so unproductive of results that other methods were sought. It was recalled that the black tar accumulated as a by-product from destructive distillation of coal had been shown to produce skin cancers when painted on mice and rabbits. Accordingly, researchers began applying tobacco smoke condensate on the backs of mice. Condensate was obtained by puffing cigarettes mechanically and passing the smoke into a cold trap where it was condensed into a dark-colored viscous liquid. Though this is not "tar" as defined in the dictionary and does not bear any close resemblance to coal tar, the term persists.

It must be pointed out that, contrary to popular belief, tobacco smoke condensate -- or "tar" -- is not actually a material to which human smokers are exposed. Many chemical studies have shown that there are qualitative, as well as quantitative differences between laboratory condensate and fresh smoke to which humans are exposed.

Nevertheless, researchers began painting such tobacco smoke condensate on the backs of mice, generally dissolved in some solvent. After persistent treatment over a long period with enormous doses, neoplasms did appear on the skins of these mice. This method of testing was seized upon by numerous other investigators with many variations of technique, different species and strains of animals and varying results. Some of these experiments were not reproducible. Chemists fractionated the tars and by the proliferating techniques of chromatography identified literally thousands of chemical components of cigarette smoke condensate with a high degree of reliability. Probably no other complex mixture in the human environment has ever been so thoroughly analyzed.

I was critical of this development from the start. A "typical" human smoker draws a puff of air through his cigarette. This generates a cloud of smoke that enters his mouth within a fraction of a second following formation, remains in his oral cavity for another fraction of a second and then is drawn into the lung diluted with five to ten volumes of air.

During this short interval, rapid physical and chemical changes are taking place. The tiny liquid droplets that constitute the blue cloud are growing in size by coalescence, which influences deposition in the lung. Numerous compounds in the aerosol are polymerizing, interacting, combining, breaking down, and otherwise changing. If such a mixture enters a cold trap, the most readily condensed substances wil be deposited as a thick liquid but other phases of the smoke escape into the air.

This situation complicates the design of devices for exposing animals to inhalation of smoke comparable in physical and chemical properties to that inhaled by human smokers. The best we can do is to design devices that will produce the smoke mechanically under conditions approximating those attained by humans and get it to the animal's lung at a comparable age and in monitored dosages.

These considerations led me to characterize the skin-painting of mice with cigarette smoke condensate as applying the "wrong material in the wrong form, in the wrong dosage, to the wrong tissue of the wrong animal." I still hold the same view.

To the best of my knowledge, to date no one has produced the type of lung cancer that is associated with human smoking by exposing experimental animals to inhalation of fresh, whole smoke. The considerations, experiences, trials, tests and findings of twenty-eight years that have been described in extreme brevity will, I hope, make it clear why The Council is now emphasizing study of the "constitutional" diseases as such on a basic level with the help of new research tools. I have tried also to show why dogmatic positions are inappropriate and restrictive in the present state of knowledge.

CURRICULUM VITAE OF DR. ROBERT C. HOCKETT

Dr. Robert C. Hockett, Research Director of The Council for Tobacco Research - U.S.A., Inc., 110 East 59th Street, New York, New York 10022, was born in Fayette, Missouri on July 1, 1906.

A graduate of the Ohio State University, Dr. Hockett pursued graduate study in the same institution and received the Ph.D. degree in chemistry there in 1929. As a National Research Council Fellow in Chemistry, he was a guest scientist at the National Institute of Health, U.S. Public Health Service from 1929 to 1931 and then Associate Chemist on the Institute staff from 1931 to 1935.

In 1935 he joined the faculty of the Massachusetts Institute of Technology as Assistant Professor of Chemistry, becoming Associate Professor in 1941.

In 1943 he was granted a leave of absence from M.I.T. to serve as Scientific Director of the Sugar Research Foundation, Inc., which position he occupied until 1952. He has also been Visiting Professor at the Universities of Illinois and North Carolina.

From 1952 until 1954 he served as a consultant to industrial firms on problems relating to foods, nutrition, pharmaceuticals, fermentations and sponsored research.

In 1954 he joined the present Council for Tobacco Research's predecessor organization - the Tobacco Industry Research Committee - as an Associate Scientific director.

Dr. Hockett is a Fellow of the American Academy of Arts and Sciences, the New York Academy of Sciences, the American Public Health Association, the Royal Society of Arts, and the American Institute of Chemists. He holds membership in the American Chemical Society, in which he has served as an Alternate Councilor, member of the Nomenclature Committee, Vice-Chairman of the Division of Carbohydrate Chemistry in 1944, Chairman in 1945 and 1946, and Secretary-Treasurer from 1956 to 1960. He also holds membership in the American Society of Biological Chemists, American Association for the advancement of Science, Friends of the World Health Organization, Royal Society of Health, and the Phi Beta Kappa Associates.

He has served as a Lecturer in Switzerland for the American-Swiss Foundation for Scientific Exchange. member of the Food Industries' Advisory Committee to the Nutrition Foundation, Inc., Collaborator to the United States Department of Agriculture, member of the Advisory Committee for Advances in Carbohydrate Chemistry, as Associate to the State Department's Committee for Interamerican Scientific Publication and member of the Sugar Advisory Committee to the Administrator of the Production and Marketing Act, U.S. Department of Agriculture.

He is author of numerous research papers on the chemistry of carbohydrates, of articles and lectures on nutrition and public health, of many reviews and summaries on tobacco and health research and contributor to a book, Beet Sugar Economics.

Relationships Between Family Smoking Habits, Individual Differences in Personality, and the Smoking Behavior of College Students

By

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Professor of Psychology
Director, Center for Research in
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I am Charles D. Spielberger, Professor of Psychology and Director of the Center for Research in Community Psychology at the University of South Florida in Tampa. I obtained my Ph.D. in Psychology at the University of Iowa in 1954, and have been a member of the psychology faculty at Duke University, Vanderbilt University and Florida State University, and directed the Doctoral Programs in Clinical Psychology at Florida State and the University of South Florida. I was also employed by the U.S. Public Health Service as a training specialist in psychology. I am certified in Clinical Psychology by the American Board of Psychologists and presently serve as Associate Editor for three major journals and on the editorial boards for a number of additional journals. I have contributed to the research literature on personality for more than 25 years, and have been interested in the association of personality and smoking behavior for the past five years.

Due to a long-standing commitment to host the Annual Meeting of the Society for Personality Assessment, in Tampa, Florida, March 11-13, 1982, in my capacity as official host for the meeting and member of the Society's National Board, I will be unable to appear in person before Congressman Waxman's Subcommittee to present the results of several recent studies in which I have been engaged, and which I believe are relevant to the Subcommittee's consideration of H.R. 4957. I have, therefore, prepared this statement for submission to the Subcommittee.

Over the past four years, I have conducted research on the relationships between family smoking habits, selected personality traits and the smoking behavior of college students. More than 1500 students have been interviewed or tested in this research. A Smoking Behavior Questionnaire (SBQ) was developed to elicit specific information about students' smoking behavior and the smoking habits of their families, and standardized personality inventories were administered to most of the participants in these studies.

The major findings that have emerged in this research are summarized in this paper. My research results are generally consistent with the increasing evidence that peer-group pressures are the single most important influence in the initiation of smoking. In this regard, it is noteworthy that the results of a related study which I am presently completing indicates that media advertising is consistently ranked near the bottom of a listing of ten factors which might be thought to influence college students to start smoking. This finding is strengthened because it was true for both females and males. (Please see Table 1, page 8.)

The second area of research reported on below provides evidence that smokers and non-smokers differ in personality, and thereby supports the hypothesis that constitutional-genetic factors contribute to the maintenance of smoking behavior.

My research results are set forth below in two sections. Relationships between family smoking habits and the initiation and maintenance of smoking behavior are reviewed in the first section. Next, relationships between important personality traits and smoking behavior are examined. Manuscripts for two papers that provide more detailed information about the research findings are included as an Appendix to this report. Both papers have been accepted for publication in scientific journals.

Family Smoking Habits and Student Smoking Behavior

For both males and females, students whose older brothers or sisters smoked were more likely to be smokers than those whose older siblings did not smoke. Older sisters appeared to have greater impact on the smoking behavior of their younger sisters than their younger brothers, whereas older brothers seem to have a similar influence on the smoking behavior of younger siblings of both sexes. These results are generally consistent with moun[illegible] evidence that peer group pressures are perhaps the single most important influence in the initiation of smoking.

A positive association was also found between the smoking behavior of college students and the smoking habits of their parents. If one or both parents smoked, their sons and daughters were more likely to be smokers than if neither parent smoked. These findings were generally consistent with results reported in eight previous investigations.

When the combined effects of the smoking habits of parents and older siblings were evaluated, older siblings appeared to have a stronger influence on the smoking behavior of younger siblings than their parents. Students whose older siblings were smokers were more likely to take up smoking than those whose older siblings were non-smokers, and parental smoking habits seemed to have no added influence on the smoking behavior of these students. Students with no older siblings, or whose older siblings were non-smokers, were less likely to be smokers themselves, but these students were more likely to take up smoking if one or both parents smoked than if neither parent smoked.

No differences were found in the smoking habits of the parents of current, occasional and ex-smokers, nor in the smoking habits of their older siblings. Thus, there was little evidence that family smoking habits influenced the maintenance of smoking behavior after the students had begun to smoke. Taken as a whole, the results indicate that family members influence students to take up smoking, but have little or no impact on the maintenance of smoking behavior.

The finding that the smoking behavior of students was more strongly associated with the smoking habits of older siblings than with parental smoking habits suggests that peer groups and other environmental influences are more important than constitutional-genetic factors in the initiation of smoking. However, the data on the relationship between personality and smoking that are discussed in the following section provide evidence that Smokers and Non-Smokers differ in personality, and thus support Eysenck's (1980) hypothesis that constitutional-genetic factors contribute to the maintenance of smoking behavior.

Personality and Smoking Behavior

Students classified as "Smokers" (Current, Occasional and Ex-Smokers) had significantly higher mean scores than Non-Smokers on the Extraversion, Neuroticism and Psychoticism scales of the Eysenck Personality Questionnaire (EPQ), and significantly lower scores on the EPQ Lie Scale. Although the observed differences between Smokers and Non-Smokers were similar for both sexes, women scored higher than men on the EPQ Neuroticism and Lie scales, and lower on Psychoticism. The differences between Smokers and Non-Smokers on all of the EPQ measures were also stronger for the female students than for the males.

Female Smokers had higher scores than Non-Smokers on the Trait Anxiety scale of the State-Trait Personality Inventory (STPI), whereas male Smokers had slightly lower anxiety scores than Non-Smokers on this scale. No significant differences were found between Smokers and Non-Smokers on the STPI Trait Curiosity and Trait Anger scales.

In evaluating the relationship between personality traits and the maintenance of smoking behavior, Ex-Smokers were found to have significantly higher STPI Curiosity scores than Current or Occasional Smokers, and female Current Smokers had lower EPQ Neuroticism scores and lower STPI Trait Anxiety scores than female Occasional and Ex-Smokers. The finding that female Current Smokers scored lower in both neuroticism and anxiety than female Occasional and Ex-Smokers was surprising, and suggested that smoking may serve primarily as a tension reducer for females who are generally higher in neuroticism than males.

Given the fact that Ex-Smokers had significantly higher Trait Curiosity scores than Current or Occasional Smokers, we may speculate that curiosity motivates Ex-Smokers to take up smoking, but this motive would be unlikely to stimulate them to continue to smoke. The finding must be replicated, however, in order to have confidence in this speculative interpretation.

While the finding that Smokers scored higher than Non-Smokers on all of the EPQ personality dimensions was consistent with results previously reported by other investigators, this was the first study to demonstrate a significant relationship between the EPQ measures and smoking behavior with an American sample. An article describing the relations between personality and smoking behaivor has been accepted for publication in the Journal of Personality Assessment. A preprint of this article is included in the Appendix.

Taken as a whole, the findings of our research provide additional support for Smith's (1970) and Matarazzo and Matarazzo's (1965) conclusions that smokers are more extraverted, neurotic and tense, and have stronger antisocial tendencies than non-smokers. The results also suggest that females, who are higher in neuroticism and trait anxiety than males, may take up smoking in order to reduce tension (Eysenck, 1980). The finding that female Current Smokers scored lower on neuroticism and trait curiosity than Occasional and Ex-Smokers further suggested that smoking may be an effective tension reducer for females who smoke regularly.

Conclusion

The research on family smoking habits reviewed above is consistent with existing evidence that peer pressures are the single most important influence in the initiation of smoking. The research on personality and smoking suggests that constitutional-genetic factors associated with personality differences contribute to the maintenance of smoking behavior.

March 8, 1982

Charles D. Spielberger

Charles D. Spielberger

Smoking Behavior

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3. Smith, G. M. Personality and smoking: A review of the empirical literature. In: W. A. Hunt (Ed.), Learning mechanisms and smoking. Chicago: Aldine, 1970.

Table 1

Rank Order, Means and Standard Deviations of Factors Reported by Male and Female College Students to Influence Them to Begin Smoking

	Females (N=294)		Males (N=130)	
Reason for Beginning to Smoke	Rank	$\bar{X}$ SD	Rank	$\bar{X}$ SD
See if I would enjoy it	1	2.61 0.95	1	2.52 0.92
Most friends smoke	2	2.52 1.14	3	2.41 1.16
Try something new	3	2.44 0.97	4	2.32 0.92
Thought it was satisfying because other people smoke	4	2.37 0.98	2	2.48 0.98
Made me feel more relaxed in social situations	5	2.14 1.05	5	2.02 1.08
Parents seemed to enjoy smoking	6	1.58 0.89	7	1.53 0.85
Did not want to refuse friends	7	1.51 0.89	6	1.55 0.87
Older siblings enjoyed smoking	8	1.48 0.83	9	1.46 0.83
Media advertisements	9	1.46 0.73	8	1.48 0.74
Parents disapproved - show independence	10	1.32 0.73	10	1.31 0.69

APPENDIX

Scientific Papers

1. Spielberger, C.D., Jacobs, G.A., Crane, R.S. & Russell, S.F. On the relation between family smoking habits and the smoking behavior of college students. International Review of Applied Psychology, In press, 1982.
2. Spielberger, C.D. & Jacobs, G.A. Personality and smoking behavior. Journal of Personality Assessment, In press, 1982.

PROFESSIONAL RESUME

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May, 1981

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B.S.	Georgia Tech	(Chemistry),	1949
B.A.	University of Iowa	(Psychology),	1951
M.A.	University of Iowa	(Psychology),	1953
PH.D.	University of Iowa	(Psychology),	1954

Major Research and Professional Interests:

Stress, anxiety and anger; test anxiety, personality and learning;
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Professional Certification:

Diplomate in Clinical Psychology, American Board of Professional Psychology, 1961

Licensed Psychologist: Tennessee, Florida

Professional Employment and Experience:

Professor of Psychology and Director, Center for Research in Community Psychology, University of South Florida, September, 1977 - Present.

Professional Employment and Experience (Continued):

Fellow, Netherlands Institute for Advanced Study, Wassenaar, The Netherlands, 1979-1980. (on sabbatical)

Professor of Psychology and Director, Doctoral Program in Clinical and Community Psychology, University of South Florida, September, 1972 - 1977.

Professor of Psychology, Florida State University, 1967-1972; Director, Doctoral Training Program in Clinical Psychology, (January, 1967 - September, 1970). FSU London Study Center, January-June, 1972.

Training Specialist in Psychology (GS-15), National Institute of Mental Health, Bethesda, Maryland, 1965 - 1967.

Professor of Psychology, Vanderbilt University, 1963 - 1967 (On leave at NIMH 1965 - 1967.

Assistant Professor to Associate Professor of Psychology, Duke University, 1955 - 1963.

Assistant Professor of Medical Psychology and Staff Psychologist, Psychiatric Outpatient Clinic, Department of Psychiatry, Duke University Medical Center, 1955 - 1958.

Clinical Psychology Intern (USPHS Fellow), Worcester State Hospital, Worcester, Massachusetts, 1954 - 1955.

Graduate Student, Department of Psychology, University of Iowa, 1951 - 1954. Research Assistant to J. S. Brown and I. E. Farber, 1951 - 1952; Psychometrist, Department of Medical Psychology, 1952 - 1953; Counselor, Student Counseling Office (USPHS Fellow), 1953 - 1954.

Military Service:

United States Navy Reserve, Commander, USNR-R, 1105/523-617.
Active Duty (3/45-8/46) as Electronics Technician.
Reserve Duty (8/46-6/79) in Navy Electronics, Training, Research, Systems Analysis, and Management Assistance Programs.
Retired Reserve: June 30, 1979.

Professional Honors and Offices:

Distinguished Scholar Award, University of South Florida, 1973 (First Recipient)

Professional Honors and Offices

Fellow, American Psychological Association, Divisions 8, 12, 13 and 27; Member, Division 3

- Elected Member, APA Committee on Accreditation, 1967-70
- Chairman, 1969-70
- Member, APA Education and Training Board, 1969-70
- Chairman, National Advisory Committee, APA Visiting Psychologist Program, 1967-70
- Council Monitor for the APA Committee on International Relations in Psychology, 1976-78. Council Liaison, 1978-79.

Award for DISTINGUISHED CONTRIBUTIONS TO PSYCHOLOGY THROUGH THEORY AND RESEARCH, Florida Psychological Association, 1977.

APA Division 27: Community Psychology

- President, 1974-75
- Chairman, Division Committee on Research and Scientific Affairs, 1969-73
- Chairman, National Program Committee, 1973-74
- Member, Executive Council, 1973 - Present

Elected to APA Council of Representatives, 1976-78

Southeastern Psychological Association:

- President, 1972-73
- Member, Executive Council, 1971-74
- Secretary-Treasurer, 1965-66
- Chairman, Division I (Universities and Colleges) 1970-71
- Chairman, Program Committee, 1971-72
- Chairman, Elections Committee, 1973-74
- Chairman, Awards Committee, 1977-78

Interamerican Society of Psychology

- Vice President for North America (U.S. and Canada), 1976-1979
- Co-Chairperson, XVI Interamerican Congress, Miami, 1976
- National Chairman for the United States, 1979-present
- Executive Secretary for the United States and Canada, 1979-present

Professional Honors and Offices (Continued):

Honorable Mention for Outstanding Research,
American Personnel and Guidance Association, 1963

Member, Juvenile Problems Research Review Committee,
National Institute of Mental Health, 1971-75

Psi Chi, The National Honor Society in Psychology
Southeastern Vice President, 1976-1980
Member National Council, 1976-present
National President and Chairman, Psi Chi National Council, 1980-present

Other Professional Affiliations:

Fellow: Sigma Xi
Fellow: American Association for the Advancement of Science
Fellow: Society for Personality Assessment
Member: American Academy of Political and Social Science
Member: American Educational Research Association
Member: American Association of University Professors
Member: Midwestern and Florida Psychological Association
Member: Psychonomics Society
Member: Society for Psychophysiological Research
Member: International Association of Applied Psychology
Member: Advisory Board, International Law Enforcement Stress Association
Member: International Advisory Committee, Second International Conference on Psychological Stress and Adjustment in Times of War and Peace, Jerusalem, Israel, June 9-23, 1978.
Member: International Council of Psycholgists

Research and Training Grants:

Principal Investigator: "Improving the Academic Adjustment of Anxious College Freshmen". NIMH Mental Health Project Grant (OM-362), 1959-1962, $73,000 (With H. Weitz).

Principal Investigator: "Mediating Processes in Verbal Conditioning". NIMH/NICHD Research Grant (MH-07446/HD 0947), 1963-1966, $61,000.

Research and Training Grants (Continued):

Program Director: NIMH Clinical Psychology Training Grant. Florida State University, 1968-1973, $574,000.

Co-Investigator: "A Program of Research on Anti-social Behavior and Violence". NIMH Research Grant (MH-1848), With E.I. Megargee and J. Hokanson, 1970-1973, $322,000.

Co-Director: (With Dr. Mitchell Silverman, Criminal Justice Program): "Psychological Assessment of Police Candidates". State-Related Grant Project M-37. January 1, 1974-June 30, 1975, $17,500.

Project Director: "Standards for the Selection of Police Officers". Law Enforcement Assistance Administration (75-DF-04-0026). April 1, 1975-March 31, 1977, $15[illegible],356.

Principal Investigator: "Improvement in the Speed of Complex Learning Through Reduction in Test Anxiety". Advanced Research Projects Agency, Department of Defense. March 16, 1977-May 31, 1979, $121,198.

Principal Investigator: An Exploratory Study of the Role of Anger and Irritability in Cardiovascular Disease. R.J. Reynolds Industries, Inc. October 15, 1977-August 15, 1981, $8,600.

Project Director: "Standards for the Selection of Police Officers". Florida Department of Criminal Law Enforcement (P-78-A2-21-CA01/79-AA-21-CA01). April 1, 1978-March 31, 1981, $146,046.

Principal Investigator: "The Origins and Correlates of Smoking Behavior". Jacob & Medinger. July 1, 1978-July 31, 1981, $59,368.

Principal Investigator: "Training Police Officers to Cope with Stress". Law Enforcement Assistance Administration, U.S. Department of Justice. July 1, 1979-March 31, 1981, $29,700.

Scientific Conferences and Institute Grants:

Scientific Director (With Prof. I. G. Sarason): Advanced Study Institute on "Stress and Anxiety in Modern Life", Murnau-Am-Staffalsee, West Germany, June 10-16, 1973. Supported by a grant from the Scientific Affairs Division, North Atlantic Treaty Organization. 950,000 Belgian Francs (approximately $25,000), with a supplementary grant from the Max Planck Institute, Munich, of 1,500 Deutsch Marks.

Scientific Director (With Prof. I. G. Sarason): International Conference on "Empirical Approaches to Stress and Anxiety", Voksenasen-Oslo, Norway, June 29-July 3, 1975. (Oiginally scheduled to be held in Athens, Greece, September 3-6, 1974, but postponed because of the Cyprus situation). Supported by a grant from the Scientific Affairs Division, North Atlantic Treaty Organization. 500,000 Belgian Francs (approximately $15,000), with a supplementary grant from Hoffman-LaRoche Pharmaceuticals of $3,000.

Scientific Director (With Prof. I. G. Sarason): Advanced Study Institute on "Coping With Stress", Urbino, Italy, July 10-17, 1976. Supported by a grant from the Scientific Affairs Division, North Atlantic Treaty Organization. 720,000 Belgian Francs (approximately $20,000).

Conference Chairman: National Conference on "Standards for the Selection of Law Enforcement Officers". National Training Academy, Federal Bureau of Investigation, Quantico, Virginia, October 26-29, 1976. Law Enforcement Assistance Administration, U. S. Department of Justice (7-0061-J-LEAA), $7,126. In addition, room and board for all Conference speakers and participants were provided by the FBI Academy.

Scientific Director: (With T. G. Sarason and W. D. Froehlich): NATO Advanced Study Institute on "Environmental Stress, Life Crises, and Social Adaptation", Cambridge, England, August 9-16, 1978. 918,000 Belgian Francs (approximately $25,000).

Conference Chairman: National Workshop on the Selection of Law Enforcement Officers. National Training Academy, Federal Bureau of Investigation, Quantico, Virginia, May 29-June 1, 1979. Law Enforcement Assistance Administration, U. S. Department of Justice (78-DF-AX-0194) $34,599.00. In addition, room, meals and local transportation for all conference speakers and participants are being provided by the FBI Academy.

Conference Director (With P. B. Defares):
Dutch National Conference on "Stress and Anxiety", Netherlands Institute for Advanced Study, February 7-9, 1980

NIAS International Conference on "Stress and Anxiety", June 19-21, 1980

Both conferences were held at the Netherlands Institute for Advanced Study, Wassenaar. Supported by grants from the Dutch Ministry of Education (20,000 Dutch Gelders) and the Netherlands Heart Foundation (12,000 Dutch Gelders).

Editorial Appointments:

Editor: Current Topics in Clinical and Community Psychology, Academic Press, 1968-1972.

Founder and Editor: American Journal of Community Psychology, Plenum Publishing Corp., New York, N.Y. 1971-September, 1976. Associate Editor, 1976-present.

Associate Editor: International Review of Applied Psychology, 1979-present.

Associate Editor: Personality and Individual Differences, 1981-present.

Consulting Editor: The Series in Clinical and Community Psychology, Hemisphere/ Wiley, 1972-1980; McGraw-Hill/Hemisphere, 1981-

General Editor: The Centennial Psychology Series, Praeger, A Division of Holt, Rinehart and Winston, CBS Inc., 1981-present.

Senior Editor: Advances in Personality Assessment, LEA, 1981-present. (With James Butcher.)

Editorial Appointments: Member of Editorial Board:

Archiv fur Psychologie, 1978-Present
Community Mental Health Review, 1975-Present
Contemporary Psychology, 1971-1974
Journal of Abnormal Psychology, 1971-1973
Journal of Behavioral Assessment, 1980-Present
Journal of Behavioral Medicine, January, 1979-Present
Journal of Educational Psychology, May, 1976-Present
Journal of Fluency Disorders, 1974-1979
Journal of Personality, 1972-1975
Journal of Personality Assessment, November, 1976-Present
Psychological Monographs, 1962-1966

Occasional Editorial Reviewer for:

American Psychologist
American Jour. of Psychology
British Journal of Psychology
Canadian Journal of
  Behavioral Science
Community Mental Health Journal
Cognitive Therapy & Research
Journal of Abnormal Child Psychology
Journal of Applied Behavior Analysis

Journal of Gerontology
Journal of Motor Behavior
Psychological Bulletin
Journal of Personlity and
  Social Psychology
Journal of Verbal Learning
  and Verbal Behavior
Multivariate Behavioral Research
Psychological Reports

Occasional Editorial Reviewer for (Continued):

- Journal of Clinical Psychology
- Journal of Consulting and Clinical Psychology
- Jour. of Cross-Cultural-Psychology
- Journal of Educational Measurement
- Psychological Review
- Psychonomic Science
- Science
- Journal of Experimental Research in Personality

Consulting Appointments During the Past 10 Years (1969-1979):

Consultant on Training and Research in Clinical Psychology, Veteran's Administration, Southeastern Region, Various Hospitals and Clinics, 1956-Present.

Bay Pines VA Hospital, St. Petersburg, Florida, 1967-Present

Attending Clinical Psychologist, Psychiatric Service, Durham, North Carolina VA Hosptial, 1956-1963

Research Consultant, VA Outpatient Clinics, St. Petersburg, Fla, 1974-Present

Consultant, Psychological Services, Federal Correctional Institution, Tallahassee, Florida, 1967-Present

Consultant, Southern Regional Education Board, Advisory Committee on Community-Clinical Psychology, 1972-1975

Consultant, Regional Flight Surgeon (Atlanta Office), Federal Aviation Administration, 1973-1977

Research Consultant, Department of Defense, Advance Research Projects Agency, Arlington, Virginia, 1976-1978

Professional Publications

Books, Chapters, Monographs and Major Articles:

1. Spielberger, C.D. The role of awareness in verbal conditioning. In C.W. Eriksen (Ed.), Behavior and Awareness, Durham: Duke University Press, 1962, pp. 73-101. Also published in J. Personality, 1962, 30, Supplement.

2. Spielberger, C.D., Weitz, H. and Denny, J.P. Group counseling and the academic performance of anxious college freshmen. Journal of Counseling Psychology, 1962, 9, 195-204. (Awarded certificate by American Personnel & Guidance Association as one of the outstanding research publications on counseling in 1962).

Books, Chapters, Monographs and Major Articles (Continued):

3. Spielberger, C.D. and Weitz, H. Improving the academic performance of anxious college freshmen: A group counseling approach to the prevention of underachievement. Psychol. Monographs, 1964, 78, 1-20 (Whole No. 590)

4. Spielberger, C.D. Theoretical and epistemological issues in verbal conditioning. In S. Rosenberg (Ed.), Directions in Psycholinguistics, New York: Macmillan, 1965, 149-200.

5. Spielberger, C.D. and DeNike, L.D. Descriptive behaviorism versus cognitive theory in verbal operant conditioning. Psych. Review, 1966, 73, 306-326.

6. Spielberger, C.D. (Ed.) Anxiety and Behavior, New York: Academic Press, 1966.

7. Chapter 1: Theory and research on anxiety, pp. 3-20
8. Chapter 14: The effects of anxiety on complex learning and academic achievement, pp. 361-398.

9. Spielberger, C.D. A mental health consultation program for small communities with limited professional resources. In E.L. Cowen, E.A.Garner, and M. Zax (Eds.), Emergent Approaches to Mental Health Problems; New York: Appleton-Century-Crofts, 1967, 214-236.

10. Spielberger, C.D., Masterton, R.B. and Fox, R. (Eds.), Contributions to General Psychology. New York: The Ronald Press Co., 1968.

11. Spielberger, C.D., Masterton, R.B. and Fox, R. Student Workbook for Principles of General Psychology, Third Edition, by G.A. Kimble and N. Garmezy. New York: Ronald Press Co., 1968. (Revised, 1970)

12. Spielberger, C.D. (Ed.) Current Topics in Clinical and Community Psychology. New York: Academic Press, Inc.

13. Volume 1, August, 1969
14. Volume 2, December, 1970
15. Volume 3, August, 1971

16. Spielberger, C.D. The effects of anxiety on computer assisted learning. In Contributions of Behavioral Science to Instructional Technology: The Affective Domain, Vol. 1. Washington, D.C.: Communications Service Corp., 1970, pp. 39-63. Paper presented at a symposium on "The Affective Domain of Learning", sponsored by Teaching Research, A Division of the Oregon State System of Higher Education, March, 1969.

Books, Chapters, Monograhs and Major Articles (Continued):

17. Iscoe, I. and Spielberger, C.D. (Eds.) Community Psychology: Perspectives in Training and Research. New York: Appleton-Century-Crofts, 1970.

18. Chapter 1: (Iscoe & Spielberger): The emerging field of community psychology, pp. 3-16

19. Chapter 15: (Spielberger & Iscoe): The current status of training in community psychology, pp. 227-246

20. Spielberger, C.D., Lushene, R.E. and Gorsuch, R.L. The State-Trait Anxiety Inventory (Test Manual). Palo Alto, California: Consulting Psychologist Press, 1970.

21. Spielberger, C.D. Trait-State Anxiety and Motor Behavior. Journal of Motor Behavior, 1971, 3, pp. 265-279

22. Spielberger, C.D., Gonzalez-Reigosa, F., Martinez-Urrutia, A., Natalicio, F.S. and Natalicio, D.S. Development of the Spanish Edition of the State-Trait Anxiety Inventory. Interamerican Journal of Psychology, 1971, 5, pp. 145-158

23. Gaudry, E. and Spielberger, C.D. Anxiety and Academic Achievement. Sydney: John Wiley Australasia, 1971.

24. Spielberger, C.D. (Ed.) Anxiety: Current Trends in Theory and Research. Vol. 1 & 2, New York: Academic Press, 1972. Based on two symposia on this subject held in Tallahassee in 1970.

25. Chapter 1: Current Trends in Theory and Research on Anxiety, pp. 3-19
26. Chapter 2: Anxiety as an Emotional State, pp. 23-49
27. Chapter 14: Conceptual and Methodological Issues in Anxiety Research, pp. 481-493.

28. Spielberger, C.D., O'Neil, H.F., Jr. and Hansen, D.N. Anxiety, Drive Theory, and Computer-Assisted Learning. In B.A. Maher (Ed.) Progress in Experimental Personality Research, Vol. 6, 1972, pp. 109-148.

29. Spielberger, C.D. and Iscoe, I. Graduate education in community psychology. In S.E. Golann & C. Eisdorfer (Eds.) Handbook of Community Psychology. New York: Appleton-Century-Crofts, 1972, pp. 909-920.

30. Spielberger, C.D. Manual for the State-Trait Anxiety Inventory for Children. Palo Alto: Consulting Psychologist Press, 1973.

31. Spielberger, C.D. The measurement of state and trait anxiety: Conceptual and methodological issues: In L. Levi (Ed.), Emotions: Their parameters and measurement. New York: The Raven Press, 1975, pp. 713-725.

32. Spielberger, C.D. and Sarason, I.G. (Eds.), Stress and anxiety, Vol. 1. Washington, D.C.: Hemisphere/Wiley, 1975.

33. Chapter 5: (Spielberger). Anxiety: State-trait-process, pp. 115-151.

Books, Chapters, Monographs and Major Articles (Continued):

34. Sarason, I.G. and Spielberger, C.D. (Eds.), Stress and anxiety, Vol. 2. Washington, D.C.: Hemisphere/Wiley, 1975.

35. Zuckerman, M. and Spielberger, C.D. (Eds.), Emotions and anxiety: New concepts, methods and applications. New York: LEA/WILEY, 1976.

36. Chapter 10: (Spielberger, Anton, and Bedell). The nature and treatment of test anxiety, pp. 317-345.

37. Sarason, I.G. and Spielberger, C.D. (Eds.), Stress and anxiety, Vol. 3. Washington, D.C.: Hemisphere/Wiley, 1976.

38. Spielberger, C.D. Stress, anxiety and cardiovascular disease. Journal of the South Carolina Medical Association, 1976, Supplement, 15-22.

39. Spielberger, C.D. and Diaz-Guerrero, R. (Eds.), Cross-cultural anxiety. Washington, D.C.: Hemisphere/Wiley, 1976.

40. Chapter 1: (Spielberger). The nature and measurement of anxiety, pp. 3-12.

41. Chapter 2: (Spielberger and Sharma). Cross-cultural measurement of anxiety, pp. 13-25.

42. Chapter 3: (Biaggio, Natalicio and Spielberger). The development and validation of an experimental Portuguese form of the State-Trait Anxiety Inventory, pp. 29-40

43. Chapter 6: (Bauermeister, Forastieri and Spielberger). Development and validation of the Spanish form of the State-Trait Anxiety Inventory for Children (IDAREN), pp. 69-85.

44. Spielberger, C.D. State-trait anxiety and interactional psychology. In D. Magnusson and N.S. Endler (Eds.), Personality at the crossroads: Current issues in interactional psychology. New York: LEA/WILEY, 1977, pp. 173-183.

45. Spielberger, C.D., Lushene, R.E., and McAdoo, W.G. Theory and measurement of anxiety states. In R.B. Cattell and R.M. Dreger (Eds.), Handbook of modern personality theory. New York: Hemisphere/Wiley, 1977, pp. 239-253.

46. Iscoe, I., Bloom, B., and Spielberger, C.D. (Eds.), Community Psychology in Transition. Washington, D.C.: Hemisphere/Wiley, 1977.

47. Chapter 1: (Iscoe & Spielberger). Community Psychology: The historical context, pp. 3-17.

48. Chapter 33: (Spielberger & Iscoe). Reflections on the Austin Conference: Community psychology in transition, pp. 315-327.

Books, Chapters, Monographs and Major Articles (Continued):

49. Spielberger, C.D. Computer-based research on anxiety and learning: An overview and critique. In J. Sieber, S. Tobias, and H.F. O'Neil (Eds.), *Anxiety, learning and instruction*. New York: LEA/WILEY, 1977, pp. 119-132.

50. Spielberger, C.D. Anxiety: Theory and research. In B.B. Wolman (Ed.), *International encyclopedia of neurology, psychiatry, psychoanalysis and psychology*, 1977, pp. 81-84.

51. Spielberger, C.D. and Sarason, I.G. (Eds.), *Stress and Anxiety*, Vol. 4, New York: Hemisphere/Wiley, 1977.

52. Spielberger, C.D. and Jacobs G.A. Stress and anxiety during pregnancy and labor. In L. Carenza, P. Pancheri, and L. Zichella (Eds.), *Clinical psychoneuro-endocrinology in reproduction*. New York: Academic Press, 1978, pp. 261-269.

53. Spielberger, C.D., Kling, J.K., and O'Hagan, S.E. Dimensions of psychopathic personality: Antisocial behaviour and Anxiety. In R. Hare and D. Schalling (Eds.), *Psychopathic Behaviour: Approaches to Research*. New York: Wiley, 1978, pp. 23-46.

54. Spielberger, C.D. and Sarason, I.G. (Eds.), *Stress and Anxiety*, Vol. 5. New York: Hemisphere/Wiley, 1978.

Chapter 9: (Spielberger, Gonzalez, Taylor, Algaze and Anton). Examination stress and test anxiety, pp. 167-191.

55. Spielberger, C.D. (Ed.), *Police Selection and Evaluation*. New York: Praeger Publishers, A Division of Holt, Rinehart and Winston, CBS Inc., 1979.

56. Chapter 2: (Spielberger, Ward & Spaulding). A model for the selection of law enforcement officers, pp. 11-29.

57. Chapter 14: (Spielberger, Spaulding, Jolley & Ward). Selection of Effective Law Enforcement Officers: The Florida Police Standards Research Project, pp. 231-251.

58. Speilberger, C.D. *Understanding Stress and Anxiety*. New York: Harper & Row, 1979.

59. Spielberger, C.D. (Ed.), *Police Selection and Evaluation: Issues and Techniques*, 1979. New York: Praeger Publishers.

60. Chapter 2: (Spielberger, Ward & Spaulding). A model for the selection of law enforcement officers, pp. 11-29.

61. Chapter 14: (Spielberger, Spaulding, Jolley & Ward). Selection of Effective Law Enforcement Officers: The Florida Police Standards Research Project, pp. 231-251.

Books, Chapters, Monographs and Major Articles (Continued):

62. Spielberger, C.D. *Preliminary Manual for the State-Trait Personality Inventory (STPI)*. Tampa, Florida: Center for Research in Community Psychology, University of South Florida, 1979.

63. Speilberger, C.D. (Ed.), *Proceedings of the national workshop on the selection of law enforcement officers.* (Monograph Series Three: No. 3). Human Resources Institute, University of South Florida, 1979.

64. Chapter 13: (Spielberger, Spaulding & Ward). The development of a psychological test battery for use in statewide police selection: The Florida Police Standards Research Project.

65. Spielberger, C.D., Anton, W.D., and Barker, L.R. *The effects of stress and anxiety on complex learning and performance for college students and Navy and Air Force recruits*. (Monograph Series Three: No. 2). Human Resources Institute, University of South Florida, 1979.

66. Spielberger, C.D., and Barker, L.R. *The relationship of personality characteristics to attrition and performance problems of Navy and Air Force recruits.* (TAEG Report No. 75). Naval Training Center, Orlando, Florida: Training Analysis and Evaluation Group, 1979.

67. Spielberger, C.D. and Jacobs, G.A. Emotional reactions to the stress of pregnancy and birth complications. In L. Carenza & L. Zichella (Eds.), *Emotion and Reproduction*. New York: Academic Press, 1979.

68. Spielberger, C.D., and Jacobs, G.A. Maternal emotions, life stress and obstetric complications. In L. Zichella & P. Pancheria (Eds.), *Psychoneuroendocrinology in reproduction*. Amsterdam, the Netherlands: Elsevier/North Holland Biomedical Press, 1979.

69. O'Neil, H.F., and Spielberger, C.D. (Eds.), *Cognitive and Affective Learning Strategies*. New York: Academic Press, 1979.

70. Chapter 5: (Spielberger, Gonzales & Fletcher). Test anxiety reduction, learning strategies and academic performances.

71. Sarason, I.G., and Spielberger, C.D., (Eds.), *Stress and Anxiety*. (Vol. 6). Washington, D.C.: Hemisphere/Wiley, 1979.

72. Spielberger, C.D. *Preliminary manual for the State-Trait Anger Scale*. Tampa, Florida: Center for Research in Community Psychology, University of South Florida, 1980.

73. Spielberger, C.D. *Test Anxiety Inventory: Preliminary professional manual*. Palo Alto, California: Consulting Psychologists Press, 1980.

Books, Chapters, Monographs, and Major Articles (Continued):

74. Sarason, I.G., and Spielberger, C.D. (Eds.), *Stress and Anxiety* (Vol. 7). New York: Hemisphere/Wiley, 1980.

75. Chapter 6: (Spielberger, Vagg, Parker, Donham & Westberry). The factor structure of the State-Trait Anxiety Inventory.

76. van der Ploeg, H.M., Defares, P.B., & Spielberger, C.D. *Handleiding bij de Zelf-Beoordelings Vragenlijst*. Lisse, The Netherlands: Swets & Zeitlinger, 1980.

X 77. Spielberger, C.D. *Manual for the State-Trait Anxiety Inventory (Revised)*. Palo Alto, California: Consulting Psychologists Press, 1981.

X 78. Spielberger, C.D. *Bibliography of research with the State-Trait Anxiety Inventory*. Palo Alto, California: Consulting Psychologists Press, 1981.

X 79. Spielberger, C.D. and Diaz-Guerrero, R. (Eds.), *Cross-Cultural Anxiety*, (Vol. 2). New York: Hemisphere/Wiley, In Press, 1981.

80. Chapter 1: (Spielberger & Diaz-Guerrero) Cross-cultural anxiety: An overview.

81. Chapter 2: (Hanin & Spielberger). The development and validation of the Russian form of the STAI.

Chapter 6: (Bauermeister, Collazo & Spielberger). The development and validation of the Spanish Form of the Test Anxiety Inventory: Inventario de Auto-evaluacion Sobre Examenes (IDASE).

Chapter 11: (McDonald & Spielberger). Measuring anxiety in hospitalized geriatric patients.

Chapter 12: (Laux & Spielberger). Stress, trait-state anxiety and learning: Two competing models.

X 82. Spielberger, C.D., Peters, R.A. & Frain, F. Curiosity and anxiety. In H.G. Voss & H. Keller (Eds.), *Curiosity research: Basic concepts and results*. Weinheim, Federal Republic of Germany: Beltz, 1981.

X 83. Spielberger, C.D., Sarason, I.G., and Milgram, N.A. (Eds.), *Stress and Anxiety*, (Vol. 8). (Proceedings of the Jerusalem Conference on Stress). Washington, D.C.: Hemisphere/Wiley, 1981.

X 84. Spielberger, C.D., Schulman, R.G., Spaulding, H.C., & Ward, J.C. *Police selection and performance, a comprehensive bibliography*. (Monograph Series Three: No. 4). Human Resources Institute, University of South Florida, 1981.

Books, Chapters, Monographs, and Major Articles (Continued):

X 85. Laux, L., Glanzmann, P., Schaffner, P. & Spielberger, C.D. *State-Trait-Angst-Inventar (STAI) Theoretische Grundlagen und Manual*. Weinheim, Germany: Beltz Test GmbH, 1981.

X 86. Lazzari, R., Pancheri, P., & Spielberger, C.D. *Manuale per la S.T.A.I. questionario di autovalutazine per l'ansia di stato e di tratto di Spielberger, Gorsuch e Lushene*. Firenzi (Florence), Italy: O.S. Firenzi, 1981.

? 87. Spielberger, C.D. and Vagg, P.R. (Eds.), *The Measurement and Treatment of Test Anxiety*. New York: Hemisphere/Wiley. In Press.

88. Chapter 3: (Spielberger). The Development of the Text Anxiety Inventory.

89. Chapter 8: (Spielberger, Gonzales & Vagg). Research on the Treatment of Test Anxiety: An Overview.

90, Chapter 14: (Fletcher & Spielberger). A Comparison of Cognitive Therapy and Rational Emotive Therapy in the Treatment of Test Anxiety.

91. Chapter 15: (Vagg & Spielberger). Current Status of research on the Nature, Measurement and Treatment of Text Anxiety.

? 92. Heinrich, D.L., & Spielberger, C.D. Anxiety and complex learning. In H.W. Krohne and L.L. Laux (Eds.), *Achievement, Stress, and Anxiety*. New York: McGraw-Hill/Hemisphere, in press.

Journal Articles and Book Reviews:

1. The effects of serial position and design difficulty on recall of the Bender Gestalt Test Designs. *Journal of Consulting Psychology*, 1955, *19*, 230-234. (With L.D. Goodstein, J.E. Williams, and W.G. Dahlstrom).

2. The relationship between "achievement imagery" and stuttering behavior in college males. *Proceedings of the Iowa Academy of Science*, 1955, *62*, 399-404. (With L.D. Goodstein and J.G. Martire).

3. The effects of stuttering behavior and response set on recognition thresholds. *Journal of Personality*, 1956, *25*, 33-45. Paper read at Midwestern Psychol. Assoc. Meetings, 1955.

4. Complex incidental learning as a function of anxiety and task difficulty. *Journal of Experimental Psychology*, 1958, *56*, 58-61. (With L.D. Goodstein and W.G. Dahlstrom). Paper read at Midwestern Psychol. Assoc. Meetings, 1956.

Journal Articles and Book Reviews (Continued):

5. On the relationship between manifest anxiety and intelligence. Journal of Consulting Psychology, 1958, 22, 220-224. Paper read at Midwestern Psychol. Assoc. meetings, 1957.

6. Factors in manic-depressive reactions. Diseases of the Nervous System, 1959, 20, 1-7. (With J.B. Parker, D.K. Wallace and J. Becker).

7. Manifest anxiety, intelligence, and college grades. Journal of Consulting Psychology, 1959, 23, 278. (With W.G. Katzenmeyer). Paper read at Southeastern Psychol. Assoc. Meetings, 1958.

8. Evidence of a practice effect on the Miller Analogies Test. Journal of Applied Psychology, 1961, 43, 259-263.

9. Further evidence of a practice effect on the Miller Analogies Test. Journal of Applied Psychology, 1961, 45, 126-127. (With R.N. Colver).

10. Frequency of blood types in a homogeneous group of manic-depressive patients. Journal of Mental Science, 1961, 107, 936-942. (With J. Parker and A. Theilie).

11. The effects of awareness and attitude toward reinforcement on the operant conditioning of verbal behavior. Journal of Personality, 1962, 30, 106-121. (With S.M. Levin and M. Shepard). Paper read at Midwestern Psycho. Assoc. Meetings, 1960.

12. The operant conditioning of plural nouns: A failure to replicate the Greenspoon effect. Psychological Reports, 1962, 11, 355-366. (With L.D. DeNike).

13. What is learned in verbal conditioning? Journal of Verbal Learning and Verbal Behavior, 1962, 1, 125-132. (With S.M. Levin).

14. On the relationship between manic-depressive psychosis and inner-directed character. Journal of Social Psychology, 1962, 57, 149-153. (With J. Becker and J.B. Parker).

15. The effects of manifest anxiety on the academic achievement of college students. Mental Hygiene, 1962, 46, 420-426. Paper read at Southeastern Psychol. Assoc. Meetings, 1961.

16. Conformity and achievement in remitted manic-depressive patients. J. Nerv. Ment. Dis., 1963, 137, 162-172. (With J.B. Parker and J. Becker).

17. Implicit espitemological bias and the problem of awareness in verbal conditioning: A reply to Greenspoon. Psychological Reports, 1963, 12, 103-106. (With L.D. DeNike).

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Journal of Personality Assessment, In Press

PERSONALITY AND SMOKING BEHAVIOR

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RUNNING HEAD: Personality and Smoking

Personality and Smoking Behavior

Abstract

This study investigated the relationship between selected personality measures and the initiation and maintenance of smoking behavior. The *Eysenck Personality Questionnaire* (EPQ), the *State-Trait Personality Inventory* (STPI) and a *Smoking Behavior Questionnaire* were administered to 955 students (603 females; 352 males). Smokers had significantly higher scores than Non-Smokers on the EPQ Extraversion, Neuroticism and Psychoticism scales, and lower scores on the Lie Scale. Female Smokers had higher STPI Trait Anxiety scores than Non-Smokers, whereas male Smokers had lower anxiety scores than Non-Smokers. Female Current Smokers had lower Neuroticism and Trait Anxiety scores than Occasional and Ex-Smokers, and Ex-Smokers of both sexes had higher scores on the STPI Trait Curiosity scale than Current and Occasional Smokers. It was concluded that the initiation and maintenance of smoking behavior are influenced by different personality factors.

Personality and Smoking Behavior

The diverse literature on smoking and personality was reviewed by Smith (1970), who observed that smoking was positively associated with the following personality traits: extraversion (12 of 15 studies); antisocial tendencies (17 of 19 studies); and impulsive behavior (6 of 8 studies). Smith concluded that smokers were more extraverted and have more antisocial tendencies than non-smokers, and that the evidence is "reasonably convincing" that smokers are also more impulsive. Although there was some evidence that smoking was associated with neuroticism and anxiety, Smith considered these findings either inconsistent or based on too few studies to draw meaningful conclusions.

Matarazzo and Matarazzo have offered a somewhat different interpretation of the literature on smoking and personality. In their view: "The results, meager and poorly supported as they are, suggest the..... presence of a slightly higher number of 'extravert', and 'neurotic', and 'tense' individuals among the smokers as compared to the non-smokers" (1965, p. 377).

The studies published since Smith's (1970) review provide further evidence that smoking is associated with extraversion and antisocial tendencies (Brackenridge & Bloch, 1972; Kanekar & Dolke, 1970; Jacobs & Spilken, 197[illegible]). Smokers also have higher scores than non-smokers on Eysenck's Psychoticism Scale (Jamison, 1978; Powell, 1977; Powell, Stewart, & Grylls, 1979), which is assumed to measure impulsive, antisocial behavior (Eysenck & Eysenck, 1975). In addition, there is growing evidence that smokers are more tense, neurotic and anxious than non-smokers as inferred from higher scores on Eysenck's Neuroticism Scale (Brackenridge & Bloch, 1972; Gupta, Sethi, & Gupta, 1975; Powell et al., 1979) and the Taylor (1953) Manifest Anxiety Scale (Houston & Schneider, 1973; Schneider & Houston, 1970), a measure of trait anxiety (Spielberger, 1966, 1972).

There are two major difficulties in interpreting the research findings on smoking and personality. First, the definition of 'smokers' and 'non-smokers' has varied from study to study. A second problem is the failure to distinguish between factors that influence people to begin to smoke and that contribute to the maintenance of smoking behavior once it has been established. In evaluating the association between personality and the *initiation* of smoking, current smokers and ex-smokers should be classified as smokers. In examining the effects of personality variables on the *maintenance* of smoking behavior, current smokers and ex-smokers must obviously be considered as separate groups. It may also be important to distinguish occasional smokers (e.g., people who smoke from time to time but *not* every day) from regular smokers, ex-smokers and non-smokers.

The primary goal of the present study was to investigate relationships between extraversion, neuroticism and psychoticism as measured by the Eysenck Personality Questionnaire (EPQ) and the initiation and maintenance of smoking behavior for American college students. Although the scales developed by Eysenck and his associates have been widely used in smoking research in England, Australia and India, no studies could be found in which these scales were used to investigate relationships between personality and smoking with American subjects. A second goal of this study was to examine the association between smoking behavior and trait anxiety as measured by the *State-Trait Personality Inventory* (STPI, Spielberger, 1979).

On the basis of previous research findings, smokers were expected to score higher than non-smokers in trait anxiety, and on the EPQ Extraversion, Neuroticism and Psychoticism scales. Since sex differences have been reported in a number of previous studies of personality and smoking behavior (see Eysenck, 1980), these relationships will be separately evaluated for males and females.

Method

Subjects

The subjects were 955 undergraduate students (603 females, 352 males) enrolled in introductory level psychology courses at a large urban state university; the median age was 19 years. These students volunteered to take part in the study and received course credit for their participation. The sample consisted of more than 70 percent of the students who were enrolled in introductory psychology at the time the study was conducted.

Test Instruments

The Eysenck Personality Questionnaire (EPQ). The EPQ (Eysenck & Eysenck, 1975), the most recent form of a series of personality inventories developed over the past 30 years by Eysenck and his colleagues (Eysenck, 1952; Eysenck, 1959; Eysenck & Eysenck, 1968), consists of 90 true-false items. The EPQ yields scores on Extraversion (E), Neuroticism (N) and Psychoticism (P), the three dimensions of Eysenck's personality theory. It also includes a Lie (L) scale.

The State-Trait Personality Inventory (STPI). The STPI was developed to measure anxiety, curiosity and anger as emotional states and personality traits (Spielberger, 1979). This 60-item self-report inventory is similar in format and conception to the State-Trait Anxiety Inventory

(Spielberger, Gorsuch, & Lushene, 1970). The three 10-item STPI trait scales require the respondents to report the frequency that they have experienced the specified personality characteristics by rating themselves on the following four-point scale: 1) Almost never; 2) Sometimes; 3) Often; 4) Almost always. Persons with high STPI scores on a particular personality trait are more prone to experience the emotional states associated with that trait than persons with low scores. Although the primary interest in the present study was in trait anxiety, the STPI Trait Curiosity and Trait Anger scales were also administered for exploratory purposes.[1]

The Smoking Behavior Questionnaire (SBQ). The SBQ is a 50-item self-report questionnaire designed to elicit specific information about students' smoking behavior and the smoking habits of their families.[2] In constructing the SBQ, a number of questionnaires used to evaluate smoking habits in previous investigations were carefully reviewed (Clausen, 1968; Horn, Courts, Taylor, & Solomon, 1959; Ikard, Green & Horn, 1969; Leventhal & Avis, 1976), and some of the items from these instruments were adapted for the present study. Information about the construction of the SBQ and the relation between student smoking behavior and family smoking habits is reported by Spielberger, Jacobs, Crane, and Russell (in press).

Procedure

In order to encourage and facilitate participation in the study by a large proportion of the students enrolled in introductory psychology courses, the testing sessions were scheduled immediately after the classes from which the students were recruited. Groups of 20 to 100 students were tested over a 10-month period. At the beginning of each testing session, the students were informed that the purpose of the study was to "learn more about the feelings and attitudes of college students". They were also informed that feedback sessions to provide additional information about the research would be scheduled near the end of the term. The students then read and signed a Consent Form, advising them that participation in the study was entirely voluntary, and would consist of completing several questionnaires.

The test instruments were administered in the following order: (a) the STPI; (b) the SBQ; (c) the EPQ. The STPI and the EPQ were given with standard instructions. On the SBQ, which inquired about past and present cigarette smoking habits, students were asked to report whether they were Current Smokers, Occasional Smokers, Ex-Smokers or Non-Smokers. The instructions defined an "Occasional Smoker" as: "Someone who smokes cigarettes from time to time but not everyday." A "Non-Smoker" was defined as someone who had never smoked, or had only experimented briefly with cigarettes, but never became a regular or Occasional Smoker.

Results

The percentages of students who indicated that they were Current Smokers, Occasional Smokers, Ex-Smokers or Non-Smokers are reported in Table 1. Current Smokers, Occasional Smokers and Ex-Smokers were classified as Smokers in determining the percentage of female and male "Smokers" reported in Table 1.

Insert Table 1 about here

Personality and the Initiation of Smoking Behavior

In evaluating the association between the personality measures and the initiation of smoking, the scores of Smokers and Non-Smokers on the

EPQ and the STPI scales were compared. The means and standard deviations for each EPQ and STPI scale are reported in Table 2. These data were evaluated in separate 2 x 2 factorial analyses of variance in which Groups (Smokers vs. Non-Smokers) and Sex were the independent variables; the results of these analyses are also reported in Table 2.

Insert Table 2 about here

The significant Groups main effects for all four EPQ scales indicated that Smokers had higher scores than Non-Smokers on Extraversion, Neuroticism and Psychoticism, and lower Lie scale scores. The significant Sex main effects for the EPQ Neuroticism, Psychoticism and Lie scales reflected the fact that the females scored higher than the males on the Neuroticism and Lie scales, and lower on Psychoticism. None of the Groups by Sex interactions were significant for any of the EPQ scales.

While the differences on the EPQ scales were in the same direction for both sexes, these differences were larger in magnitude for the females, except for Neuroticism on which comparable differences were found. In the separate analyses for each sex, the female Smokers scored significantly higher than Non-Smokers on Psychoticism and Extraversion, and significantly lower on the EPQ Lie scale. Male Smokers also had significantly lower Lie scale scores than Non-Smokers.

For the STPI scales, the only statistically significant differences were the Sex main effect and the Groups by Sex interaction for trait anxiety. These findings reflected the fact that females had higher anxiety scores than males, and that female Smokers had _higher_ anxiety scores than Non-Smokers, whereas male Smokers had _lower_ anxiety scores than Non-Smokers. No significant differences were found for either sex in the separate analyses of the STPI scales.

Personality and the Maintenance of Smoking Behavior

The mean EPQ and STPI scores of the female and male Current, Occasional and Ex-Smokers are reported in Table 3. Associations between the maintenance of smoking behavior and each personality measure were evaluated in 3 x 2 factorial analyses of variance in which Groups (Current, Occasional and Ex-Smokers) and Sex were the independent variables. Summaries of these analyses are also reported in Table 3, along with the findings in the separate analyses of the data for female and male students.

Insert Table 3 about here

There were no statistically significant Group differences in the overall analyses of the EPQ scores, but sex main effects were found for the Neuroticism and Psychoticism measures. The females scored higher on N and lower on P than males, as was noted in the preceding analyses. Occasional Smokers of both sexes also had lower Lie scores than the Current and Ex-Smokers, who were quite similar on this scale. In the separate analyses for females, the Current Smokers had lower Neuroticism scores than the Occasional and Ex-Smokers.

In the analyses of the data for the STPI scales, the statistically significant Groups effect for Trait Curiosity reflected the finding that Ex-Smokers of both sexes had higher scores on this measure than Current and Occasional Smokers. A significant Sex main effect was also found for Trait Anxiety, which indicated that females had higher anxiety scores on this scale than males, as was noted in the preceding analyses. In the separate analyses for females, the significant Groups effect for the STPI trait anxiety measure reflected the fact that Current Smokers had lower anxiety scores than Occasional and Ex-Smokers.

Discussion

In the present study, Smokers scored higher than Non-Smokers on the EPQ Extraversion, Neuroticism and Psychoticism scales, and Non-Smokers had higher Lie scores than Smokers. While these findings were generally consistent with results previously reported by other investigators (Brackenridge & Bloch, 1972; Gupta et al., 1975; Powell et al., 1979), this is the first study to obtain such relationships for an American sample.

The differences observed between Smokers and Non-Smokers on the EPQ scales were in the same direction for both sexes, but these differences were larger in magnitude for the females. The findings in the present study that females scored higher than males on the Neuroticism and Lie scales, and lower on Psychoticism, were consistent with the results reported for British subjects in the EPQ Test Manual (Eysenck & Eysenck, 1975).

In evaluating the association between the STPI scales and the initiation of smoking, it was found that female Smokers had higher anxiety scores than Non-Smokers, whereas male Smokers had lower anxiety scores than Non-Smokers. The results for the females are consistent with findings reported by several previous investigators that smokers scored higher in anxiety than non-smokers (Houston & Schneider, 1973; Schneider & Houston, 1970), and support Matarazzo and Matarazzo's (1965) conclusion that there are, "...a slightly higher number of...'neurotic', and 'tense' individuals among smokers as compared to the non-smokers" (p. 377). The findings in the present study of a positive association between smoking and anxiety for females and a negative association for males points up the importance of taking the sex of the subject into account in an investigation of the relationship between smoking and personality.

In evaluating the association between personality and the maintenance of smoking behavior, no significant differences were found for Current, Occasional and Ex-Smokers in the overall analyses of the EPQ *E*, *N* and *P* scores. However, in the separate analyses for females, the Current Smokers had *lower* scores on Neuroticism than the Occasional and Ex-Smokers. A similar trend was also found for the males, but this difference was not statistically significant, perhaps due to the fact that the magnitude of the differences were larger for the females and the male sample was smaller. Female Current Smokers also scored lower on the STPI Trait Anxiety Scale than Occasional and Ex-Smokers.

Taken together, the results of the present study suggest that females, who are higher in neuroticism and trait anxiety than males, may take up smoking in order to reduce tension (Eysenck, 1980). The finding that female Current Smokers scored lower on neuroticism and trait curiosity than Occasional and Ex-Smokers further suggests that smoking may be an effective tension reducer for those females who smoke regularly. However, an alternative explanation is that occasional smokers and ex-smokers are higher in neuroticism and trait anxiety before they begin to smoke. A longitudinal prospective study of smoking behavior will be required to evaluate these alternative interpretations of the present findings.

An interesting new finding in the present study was that Ex-Smokers of both sexes had higher scores on the STPI Trait Curiosity Scale than Current and Occasional Smokers. We may speculate that curiosity is an important factor in motivating the Ex-Smoker to take up smoking, and that this motive would be unlikely to stimulate them to continue smoking. Thus, for Ex-Smokers, curiosity may influence the initiation of smoking, but not the maintenance of smoking behavior. The finding must be replicated, however, in order to have confidence in this speculative interpretation.

Smokers of both sexes had lower scores on the EPQ Lie Scale (see Table 2), and Occasional Smokers of both sexes had lower Lie scores than Current and Ex-Smokers (See Table 3). Although the Lie Scale was originally designed to measure the tendency to dissimulate ("fake good"), recent research suggests that low scores on this scale may be associated with nonconforming and rebellious attitudes (Eysenck, 1980). Therefore, the finding in the present study that Smokers had lower Lie scores was quite consistent with Smith's (1970) observation that smoking was associated with antisocial tendencies in 17 of 19 studies. The fact that Occasional Smokers had lower Lie scores than Current or Ex-Smokers further suggests that Occasional Smokers may take up smoking as a non-conformist behavior, and may also resist pressure from their peers to be regular smokers.

Of the 425 students classified as Smokers in the present study, 116 (27%) smoked only "from time to time, but not every day", and there were critical personality differences between these Occasional Smokers and Current Smokers. Moreover, the relationships between personality and the initiation and maintenance of smoking appeared to be somewhat different for men and women. Thus, it would seem important to distinguish between current and occasional smokers, and to take sex differences into account in future investigations of personality and smoking behavior.

Taken as a whole, the results of the present study provide additional support for Smith's (1970) and Matarazzo and Matarazzo's (1965) conclusions that smokers are more extraverted, neurotic and tense, and have stronger antisocial tendencies than non-smokers. Nevertheless, perhaps the main conclusions that can be drawn from this study are that the relation between smoking and personality is exceedingly complex, and that different personality factors may influence the initiation and maintenance of smoking behavior.

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Footnotes

1. The STPI state scales were also administered as a part of the test battery, but will not be considered here because the main concern in the present paper is with the association between individual personality traits and smoking behavior.

2. A copy of the SBQ may be obtained by writing to the first author.

Table 1

Number and Percentage of Current Smokers, Occasional Smokers, Ex-Smokers and Non-Smokers

	Females		Males	
	N	%	N	%
Non-Smokers	308	51%	222	63%
Smokers	295	49%	130	37%
Current Smokers	117	19%	40	11%
Occasional Smokers	78	13%	38	11%
Ex-Smokers	100	17%	52	15%
Total Sample	603	100%	352	100%

Table 2

Means, Standard Deviations, and Analyses of Variance of the Scores of Smokers and Non-Smokers on the Eysenck Personality Questionnaire (EPQ) and the Trait Scales of the State-Trait Personality Inventory (STPI)

Personality Measures	Groups X Sex ANOVA F-Ratios Groups	Sex	Int	Females Smokers (N=260)	Non-Smokers (N=274)	F-Ratio	Males Smokers (N=121)	Non-Smokers (N=207)	F-Ratio
EPQ									
Extraversion	4.50*	1.32	0.75	13.74 4.19	14.97 4.57	4.13*	15.10 4.18	14.81 4.42	0.34
Neuroticism	5.41*	32.00***	2.13	13.12 4.83	12.51 5.34	1.97	11.01 5.93	10.44 5.25	0.82
Psychoticism	4.97*	38.35***	1.52	3.48 2.36	2.79 2.19	12.19***	4.23 2.62	4.08 2.69	0.26
Lie Scale	12.81***	7.98**	1.44	6.45 3.47	7.48 3.83	10.67***	5.83 3.22	6.68 3.71	4.39*
STPI									
T-Anxiety	1.30	10.2[illegible]***	4.90**	20.23 5.60	19.45 5.83	2.52	18.19 4.84	19.77 5.21	1.01
T-Anger	0.55	0.18	0.62	19.79 5.00	19.46 5.23	0.55	19.30 4.83	19.41 4.90	0.02
T-Curiosity	0.12	1.14	1.51	28.93 4.57	29.19 4.72	0.41	29.12 4.65	28.46 4.48	1.57

*p .05
**p .01
***p .001

Table 3

Means, Standard Deviations and Analyses of Variance of Scores on the Eysenck Personality Questionnaire and the State-Trait Personality Inventory for Female and Male Current Smokers, Occasional Smokers and Ex-Smokers

Personality Measure	Group X Sex ANOVA F-Ratios Group	Sex	Int	Females Current Smokers (N=102)	Occas. Smokers (N=66)	Ex-Smokers (N=92)	F-Ratio	Males Current Smokers (N=38)	Occas. Smokers (N=34)	Ex-Smokers (N=49)	F-Ratio
EPQ											
Extraversion	1.89	1.56	0.90	16.1 3.9	15.6 4.7	15.5 4.1	0.58	15.9 4.0	15.4 4.0	14.3 4.3	1.73
Neuroticism	2.25	14.93***	0.48	12.2 5.0	13.5 4.3	13.9 4.9	3.56*	10.5 5.8	11.6 6.6	11.0 5.7	0.27
Psychoticism	1.47	8.38**	0.13	3.7 2.4	3.6 2.4	3.1 2.2	1.66	4.5 3.1	4.1 2.7	4.1 2.2	0.35
Lie	3.20*	2.57	0.12	6.6 3.4	5.7 3.4	6.9 3.5	2.49	6.1 3.1	5.2 3.1	6.1 3.4	0.82
STPI											
Trait Anxiety	2.32	13.28***	1.73	19.0 5.3	21.1 5.8	21.0 5.6	4.17*	18.4 4.8	18.5 4.9	17.9 4.9	0.20
Trait Anger	0.61	0.36	0.35	19.5 5.4	20.0 [illegible]	20.0 4.3	0.36	19.3 5.7	20.3 4.9	19.1 4.0	0.66
Trait Curiosity	4.20*	0.11	0.14	28.9 4.7	28.0 4.3	29.6 4.6	2.42	28.8 4.5	28.1 4.8	30.1 4.6	1.80

*p .05
**p .01
***p .001

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On the Relation Between Family Smoking Habits and the Smoking Behavior of College Students

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Running Head: Family Smoking Habits and Student Smoking Behavior

Abstract

This study investigated the relationship between the smoking behavior of college students (603 females; 332 males) and the smoking habits of their parents and older siblings. Students responded to a *Smoking Behavior Questionnaire* that required them to report whether they were Current Smokers, Occasional Smokers, Ex-Smokers, or Non-Smokers; similar information was obtained about their parents and older siblings. Of the females, 49% were classified as Smokers as compared to only 37% of the males. Students whose parents or older siblings smoked were more likely to be Smokers themselves; older siblings appeared to have a greater influence on the smoking behavior of younger siblings than did their parents. No differences were found in the smoking habits of the parents or the older siblings of Current, Occasional and Ex-Smokers. Contrary to previous investigations, there was no evidence of same-sex parental modeling of smoking behavior.

On the Relation Between Family Smoking Habits and the Smoking Behavior of College Students

The controversy on smoking and health has stimulated extensive interest in identifying factors that influence the initiation and maintenance of smoking behavior. In reviews of research in this field (Matarazzo & Matarazzo, 1968; Wohlford & Giammona, 1969; Evans, Henderson, Hill, & Raines, 1979; Leventhal & Cleary, 1977, 1980), social influence variables such as parental smoking habits and peer-group pressures have been repeatedly identified with the initiation of smoking, but relatively little is known about the factors that maintain the smoking habit.

Positive relationships between the smoking habits of parents and the smoking behavior of their children have been reported in eight studies (Banks, Bewley, Bland, Dean, & Pollard, 1978; Borland & Rudolph, 1975; Clausen, 1968; Horn, Courts, Taylor, & Solomon, 1959; Merki, Creswell, Stone, Huffman, & Newman, 1970; Palmer, 1970; Salber & MacMahon, 1961; Wohlford, 1970); only one study, which was based on a very small sample of college students, failed to find any relationship between these variables (Straits & Sechrest, 1963). Although an empirical relationship between parental smoking habits and children's smoking behavior seems firmly established, it is not clear whether this relationship reflects environmental or constitutional-genetic influences (see Eysenck, 1980).

Wohlford (1970) has called attention to the importance of examining the differential impact of the smoking habits of fathers and mothers on the smoking behavior of their sons and daughters. Sons were found to be more likely to smoke if their fathers smoked in four studies that examined this relationship

(Banks et al., 1978; Horn et al., 1959; Salber & MacMahon, 1961; Wohlford, 1970), and daughters were found to be more likely to smoke if their mothers smoked in three of these studies. Since no relationship was found between the smoking habits of fathers and daughters, nor between mothers and sons, same-sex parental modeling appears to have a stronger impact than genetic factors on children's smoking behavior.

Peer group pressure is also widely recognized as a primary factor in the initiation of smoking (e.g., Eysenck, 1980; Matarazzo & Matarazzo, 1968). Evidence of peer-group influence on the initiation of smoking was reported in six studies that investigated this relationship (Banks et al., 1978; Hill, 1971; Levitt & Edwards, 1970; Matthews, 1974; Merki et al., 1970; Palmer, 1970). Leventhal and Cleary (1980) have recently suggested that peers and parents are both important sources of environmental influence in cigarette smoking, and that older siblings may be even more important than other peers in influencing adolescents to initiate smoking. Consistent with this view, Banks et al. (1978) found that junior and senior high school students whose siblings smoked were more likely to be smokers themselves.

Only one published study could be located in which the relationship between family smoking habits and the maintenance of smoking behavior was investigated. Laoye, Creswell and Stone (1972) found that secondary school students who were regular smokers were more likely to have parents and friends who smoked than students who had been regular smokers but subsequently stopped.

The goals of the present study were to investigate the influence of the smoking habits of parents and older siblings on the initiation and maintenance of smoking behavior for college students. On the basis of previous research findings, positive relationships were expected between family smoking habits and the initiation of smoking behavior, and between the smoking habits of fathers and sons, and mothers and daughters. The maintenance of smoking behavior was investigated by comparing ex-smokers with current and occasional smokers. On the basis of the findings reported by Laoye et al. (1972), current smokers were expected to be more likely than ex-smokers to have parents and older siblings who smoked.

Method

Subjects

The subjects were 955 undergraduate students enrolled in introductory level psychology courses at a large urban state university; the median age was 19 years. All students volunteered to take part in the study and received credit for their participation. The population served by the university is comprised primarily of families of low to average socio-economic status. More than 85 percent of the 24,000 students at the university are commuters, and many of them continue to reside with their parents.

The students were tested over a period of ten months. Sample I consisted of 460 students (166 males, 294 females) tested during the Winter and Spring quarters of the 1978-79 academic year; approximately 65 percent of the students enrolled in introductory psychology courses were included in this sample. Sample II consisted of 495 students (186 males, 309 females) tested during the Fall quarter of 1979; this sample included approximately 80 percent of the students enrolled in introductory psychology courses.

Construction of the Smoking Behavior Questionnaire (SBQ)

The SBQ is a 50-item self-report questionnaire designed to elicit specific information about students' smoking behavior and the smoking habits of their families. In constructing the SBQ, a number of questionnaires used to evaluate smoking habits in previous investigations were carefully reviewed (Clausen, 1968; Horn et al., 1959; Ikard, Green & Horn, 1969; Leventhal & Avis, 1976); items from these instruments were adapted for the present study.

A preliminary form of the SBQ was administered to 149 undergraduate students (52 males, 97 females) enrolled in introductory psychology courses. In responding to the questionnaire, the students were asked to indicate if they were interested in meeting in small groups to discuss reasons why college students start and continue to smoke: Each student was promised $2.00 for participating in these discussion sessions. A total of 81 students met with the investigators in small groups of 7 to 10 students. In order to permit in-depth discussion of the students' reasons for smoking or not smoking, there were separate groups for current smokers (N=27), ex-smokers (N=17), and non-smokers (N=37). The group discussions were audio tape-recorded .

On the basis of an analysis of the responses to the preliminary SBQ and a review of the audio tape, the final set of items for the form of the SBQ that was used in the present study were selected. The data presented in this study are based on the first part of the questionnaire, which inquired about the smoking habits of the students, their parents, and their older brothers and sisters.[1]

Procedures

The SBQ was administered as part of a larger test battery to groups of 20 to 100 students. In order to encourage participation in the study, most of the group-testing sessions were scheduled immediately after the psychology classes from which the students were recruited. At the beginning of each testing session, the students were required to read and then sign a Consent

Form, which informed them that participation in the study was entirely voluntary, and would consist of responding to several questionnaires. The students were then instructed to answer each questionnaire as honestly and accurately as possible. They were also informed that feedback sessions in which they could learn more about the study would be scheduled at the end of the term.[2] Prior to administering the SBQ, the following instructions were read to the subjects:

> Now, turn to the Cigarette Smoking Questionnaire...For Section 1, indicate whether you are a Current Smoker, Occasional Smoker, Ex-Smoker, or have never smoked by placing a check in the appropriate space under the column "Your Self". An "Occasional Smoker" is someone who smokes cigarettes from time to time but not everyday. Whatever your own smoking habits, be sure to also check whether your father, mother or older brother or sister is a Current, Occasional, or Ex-Smoker or has never smoked. (Students with more than one older sibling were asked to provide information about the older brother and/or sister who were the heaviest smokers.)

Results

In evaluating the relationship between family smoking habits and the initiation of smoking behavior, students classified as Current, Occasional and Ex-Smokers were considered "Smokers", and were treated as a single group in the statistical analyses. In examining the relation between the smoking habits of parents and older siblings and the maintenance of smoking behavior, the students who were classified as Current, Occasional, and Ex-Smokers were treated as separate independent groups. In all of the statistical analyses, students who reported they had never smoked were classified as "Non-Smokers".

Students who indicated they had experimented briefly with cigarettes, but had never become either regular or occasional smokers were also included in the Non-Smoker category.

The percentages of male and female students classified as Smokers or Non-Smokers in the two samples are reported in Table 1. Since these percentages were quite similar, the data for the two samples were combined. For the combined sample, the percentage of female smokers (49%) was substantially larger than the percentage of male smokers (37%); the difference between these percentages was statistically significant (X^2=12.94, df=1, $p<.001$).

Insert Table 1 about here

Family Smoking Habits and Students' Smoking Behavior

On the basis of the smoking habits of their parents, Smokers and Non-Smokers were assigned to one of the following three categories: (a) Neither parent smoked; (b) Mother or Father smoked, but not both; and (c) Both parents smoked. In Table 2, it can be noted that most students who reported that neither parent smoked were themselves Non-Smokers (females, 64%; males, 71%), and that the percentage of Smokers was higher if mother or father or both parents smoked than if neither parent smoked. In separate 3 x 2 Chi Square analyses, the relationship between parental smoking habits and the students' smoking behavior was statistically significant for females (X^2=11.03, df=2, $p<.01$), but not for males (X^2=3.57, df=2).

Insert Table 2 about here

In order to clarify the relationship between students' smoking behavior and the smoking habits of their parents, the percentages of female and male Smokers and Non-Smokers who reported that "Neither Parent Smoked" were compared in separate 2 x 2 Chi Square analyses with the percentages who reported that either mother or father smoked, or that both parents smoked. A significantly higher percentage of the females who reported that mother or father smoked ($X^2=11.33$, df=1, $p<.001$), or that both parents smoked ($X^2=6.75$, df=1, $p<.01$), were themselves Smokers. For males who reported that mother or father smoked, the percentage of Smokers (44%) was also significantly higher than for males (29%) who reported that neither parent smoked ($X^2=3.98$, df=1, $p<.05$), but not for males (35%) who reported that both parents smoked ($X^2=0.86$, df=1).

Surprisingly, the percentage of Smokers was slightly higher for students who reported that one parent smoked than for those who reported that both parents smoked, but these differences were not statistically significant for either females ($X^2=1.17$, df=1) or males ($X^2=1.95$, df=1). Since the trends for males and females in this comparison were similar, the data for the two sexes were combined and evaluated in a 2 x 2 Chi Square analysis, which approached significance ($X^2=3.06$, df=1, $p<.10$).

If there is same-sex modeling of parental smoking habits, it would be expected that the percentage of smokers would be greater among sons whose fathers smoked, and among daughters whose mothers smoked. Of the 336 females who reported that their mothers smoked, 52% were Smokers, as compared with 51% of the 414 females whose fathers smoked. For the 259 males whose fathers smoked, 38% were Smokers; 39% of the 188 males whose mothers smoked were themselves Smokers. Since the percentage of females and males who smoked was approximately the same, irrespective of whether their mothers or fathers smoked, there appears to be little evidence of same-sex modeling by children of the smoking behavior of their parents.

Of the 933 students who participated in the present study, 398 females and 223 males reported that they had older siblings, and 177 females and 80 males reported that they had both older brothers and older sisters. For the students with older siblings, it can be seen in Table 3 that the percentage of Smokers and Non-Smokers was approximately the same as for the total sample (compare column 1 in Table 3 with the data for the combined sample in Table 1).

Insert Table 3 about here

Students with older siblings were assigned to one of the following three categories: (a) Neither older brother nor older sister smoked; (b) Older brother or older sister smoked, but not both; (c) Older brother and older sister both smoked. The percentage of Smokers and Non-Smokers in each of these categories is reported in Table 3, in which it may be noted that the percentage of Smokers was highest for students with older brothers and sisters who smoked and lowest for those whose siblings were non-smokers. More than 70% of the students of both sexes whose older siblings were non-smokers were themselves Non-Smokers. Differences in the percentages of Smokers and Non-Smokers in the three categories were evaluated in 2 x 3 Chi Square analyses, and found to be significant for both females ($X^2=43.39$, df=2, $p<.001$) and males ($X^2=9.96$, df=2, $p<.01$).

The relationship between students' smoking behavior and the smoking habits of their older siblings was further evaluated in 2 x 2 Chi Square analyses. The percentage of Smokers was significantly higher for students whose older brothers *or* sisters smoked, and for students with *both* older brothers *and* sisters who smoked, than for students with older siblings who were non-smokers (*Column 3 vs. Column 2*: Females, $X^2=30.85$, df=1, $p<.001$; Males, $X^2=7.86$, df=1, $p<.01$; *Column 4 vs. Column 2*: Females, $X^2=30.39$, df=1, $p<.001$; Males, $X^2=5.63$, df=1, $p<.05$). Although the percentage of Smokers in the "brother *and* sister smoked" category was slightly higher than in the "brother *or* sister smoked" category, this difference was not statistically significant for either females ($X^2=2.71$, df=1) or males ($X^2=0.46$, df=1).

In order to evaluate the possibility that the smoking habits of older brothers and older sisters had differential influence on the smoking behavior of their younger siblings, the percentage of Smokers and Non-Smokers among students whose older siblings smoked was compared. For the 215 students whose older sisters smoked, the percentage of female Smokers (66%) was significantly higher than the percentage (46%) of male Smokers ($X^2=8.12$, df=1, $p<.01$), whereas the difference in the percentages of female (60%) and male (54%) Smokers for the 231 students whose older brothers smoked was not significant ($X^2=0.73$, df=1). Thus, older sisters seemed to have a greater impact on the smoking behavior of their younger sisters than on their younger brothers, whereas older brothers appeared to have a similar influence on the smoking behavior of younger siblings of both sexes.

In the preceeding analyses, older siblings appeared to have a greater influence on the smoking behavior of their younger siblings than did their parents. The combined influence of the smoking habits of parents and older siblings on the smoking behavior of the 933 students who participated in the present study is examined in Table 4. Evaluation of the differences in the percentages of Smokers and Non-Smokers for the six smoking categories defined by the smoking habits of parents and older siblings resulted in highly significant 2 x 6 Chi Squares for both females ($X^2=40.34$, df=5, $p < .001$) and males ($X^2=23.79$, df=5, $p < .001$). In general, the percentage of Smokers was higher for students with older siblings who were smokers (Columns 4-6), and lower for students whose older siblings (if any) were non-smokers (Columns 1-3).

Insert Table 4 about here

In order to further clarify the relationship between family smoking habits and the smoking behavior of the students in the present study, separate 2 x 3 Chi Squares were computed for students with older brothers and/or sisters who smoked (See Table 4, Columns 4-6), and for students with no older siblings who smoked (Columns 1-3). As previously noted, students whose older siblings smoked were more likely themselves to be Smokers, but parental smoking habits appeared to have no influence on their smoking behavior (females: $X^2=1.72$, df=1; males: $X^2=4.28$, df=2). In contrast, although students with no older siblings who smoked were less likely to be Smokers, these students were more likely to smoke if one or both parents smoked than if neither parent smoked (females: $X^2=15.53$, df=2, $p < .001$; males: $X^2=8.58$, df=2, $p < .01$).

Family Smoking Habits and the Maintenance of Smoking Behavior

In order to investigate the relationship between parental smoking habits and the maintenance of smoking behavior, students classified as Current, Occasional or Ex-Smokers were assigned, on the basis of the smoking habits of their parents, to one of the following three categories: (a) Neither parent smoked; (b) Mother or Father smoked, but not both; or (c) Both parents smoked. Surprisingly, as can be noted in Table 5, a higher percentage of females whose parents did not smoke were Current Smokers than was the case for students whose parents smoked. As expected, a larger percentage of the males were Current Smokers if one or both parents smoked than if neither parent smoked. However, when evaluated in 3 x 3 Chi Square analyses, these differences were not statistically significant for either females (X^2=5.72, df=4) or males (X^2=9.29, df=4).

Insert Table 5 about here

The relation between the smoking habits of older siblings and the maintenance of smoking behavior is presented in Table 6; these data are based on the 193 female and 88 male Smokers with older siblings. No significant differences were found in the percentages of female Current, Occasional and Ex-Smokers categorized on the basis of the smoking habits of their older siblings (X^2=3.07, df=4). Since the number of males with older brothers and sisters who smoked was very small, this group was combined with the males whose older brothers or sisters smoked. No significant differences were found in the percentages of Current, Occasional and Ex-Smokers for the combined group as compared with males who had neither older brothers nor sisters who smoked (X^2=4.20, df=2).[3]

Discussion

Of the 953 college students who participated in the present study, 49 percent of the females were classified as Smokers as compared to only 37 percent of the males. In commenting on recent trends in smoking behavior, Evans et al. (1979) observed: "The rate of smoking is decreasing among adult males, adult females, and male teenagers with the only increase — a noteable one — occurring among females in their teens and early twenties" (p. 204). Thus, the finding in the present study that the percentage of smokers was significantly higher for females than for males appears to be consistent with recent trends in the smoking habits of young adults.

Positive relationships were found as predicted in the present study between the smoking behavior of students and the smoking habits of their parents. If one or both parents smoked, their sons and daughters were more likely to be smokers than if neither parent smoked. These findings were generally consistent with the results reported in six previous investigations (Banks et al., 1978; Borland & Rudolph, 1975; Horn et al., 1959; Merki et al., 1970; Palmer, 1970; Salber & MacMahon, 1961). Positive relationships have also been reported for females (Clausen, 1968) and for males (Wohlford, 1970) in two additional studies.

Positive relationships were also expected between the smoking habits of fathers and sons, and of mothers and daughters, but no evidence of same-sex parental modeling was found. A possible explanation is that the college students in the present study were older than the junior and senior high school students in previous studies (Horn et al., 1959; Salber & MacMahon, 1961; Banks et al., 1970) for whom evidence of sex-linked modeling of smoking behavior was reported.[4] Thus, sex-linked parental modeling may influence some children to begin smoking at an earlier age, but this relationship may be attenuated or masked as the number of smokers increases during the high school and college years due primarily to peer group pressures. Consistent with this interpretation, the Surgeon General's report on Smoking and Health (1964) has suggested, "...that parents' influence affects the age at which children start smoking much more than it affects the ultimate taking or not taking up of the habit" (p. 370).

In the present study, students whose older brothers or sisters smoked were much more likely to be Smokers than those whose older siblings did not smoke (See Table 3). There was also some evidence that older sisters may have a greater impact on the smoking behavior of their younger sisters than their younger brothers, whereas older brothers appear to have a similar influence on the smoking behavior of younger siblings of both sexes. These results were generally consistent with Banks et al.'s (1978) finding that high school students with siblings who smoked were more likely to take up smoking than students whose siblings were non-smokers, and with the mounting evidence that peer group pressures are perhaps the single most important influence in the initiation of smoking (Eysenck, 1980; Levitt & Edwards, 1970; Matthews, 1974; Merki et al., 1970; Palmer, 1970).

When the combined effects of the smoking habits of parents and older siblings were evaluated, older siblings appeared to have a stronger influence on the smoking behavior of their younger siblings than did their parents. The students whose older siblings were Smokers were more likely to take up smoking than those whose older siblings were non-smokers, and parental smoking habits seemed to have no added influence on the smoking behavior of these students (See Table 4). Students with no older siblings, or with older siblings who were non-smokers, were less likely to be Smokers themselves, but these students were more likely to take up smoking if one or both parents smoked, than if neither parent smoked.

No differences were found in the smoking habits of the parents of Current, Occasional and Ex-Smokers in the present study, nor in the smoking habits of their older siblings. Thus, there was little evidence that family smoking habits influence the maintenance of the students' smoking behavior.

At the time the data for the present study were collected, the National Institute of Education (NIE, 1979) of the U.S. Department of Health, Education and Welfare initiated a major investigation of patterns of teenage smoking. In this study, a sample of 2639 American teenagers were interviewed by telephone to obtain information about their smoking habits and the smoking behavior of their families and friends. Although no statistical analyses were reported, the findings in the NIE study for high school students were similar in many respects to those obtained for college students in the present study. For example:

1. The data from the NIE study indicated that the percentage of girls who smoked was larger than the percentage of boys, whereas a decade earlier it was found that the percentage of boys who smoked was higher.

2. The NIE study found that if one or both parents smoked, their children were more likely to smoke, as was the case in the present study. There was also little evidence of same-sex modeling.

3. A positive relationship was found between the smoking behavior of teenagers and the smoking habits of their older siblings similar to the relationship for college students in the present study.

4. When the combined effects of the smoking habits of parents and older siblings were evaluated, older siblings had a stronger influence on their younger siblings than did their parents.

Taken as a whole, the results of the NIE study and the present investigation indicate that family members influence students to take up smoking. The fact that the smoking behavior of the students is more strongly associated with the smoking habits of their older siblings than with parental smoking habits suggests that environmental influences are more important than constitutional-genetic factors in the initiation of smoking. The findings in the present study that the percentage of smokers tended to be higher for students who reported that one parent smoked than for those who reported that both parents smoked provides further support for this interpretation.

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Footnotes

1. A copy of the SBQ may be obtained by writing to the first author.

2. Approximately two weeks before the end of the term, the students were notified of the dates and times for the feedback sessions and were encouraged to attend.

3. There was no evidence of a relationship between the combined influence of the smoking habits of parents and older siblings on the maintenance of smoking behavior of the students who participated in this study, as evaluated in 3 x 6 Chi Square analyses similar to those computed for Table 4.

4. Wohlford (1970) reported a positive relationship between the smoking habits of fathers and sons for college students, but not for mothers and daughters. It should be noted, however, that this relationship was based on a marginally significant correlation ($p < .06$) in "cigarette smoking frequency" for fathers and sons, rather than an evaluation of a correspondence in the prevalence of smoking behavior. Differences in Wohlford's findings and those of the present study might also reflect changes in the smoking patterns of young adults that have occurred over the past decade.

Table 1

Percentage of Male and Female Smokers and Non-Smokers in Two Samples of Undergraduate College Students

	Sample I	Sample II	Combined Sample
Total Number	460	495	955
Smokers	43%	46%	45%
Non-Smokers	57%	54%	55%
Females (N)	294	309	603
Smokers	48%	50%	49%
Non-Smokers	52%	50%	51%
Males (N)	166	186	352
Smokers	35%	39%	37%
Non-Smokers	65%	61%	63%

Table 2

Relationship Between Parental Smoking Habits and the Smoking Behavior of College Students

	Number Of Subjects	Neither Parent Smoked	Mother Or Father Smoked	Both Parents Smoked
Females (N)	603	118	219	266
% Smokers	49%	36%	55%	50%
% Non-Smokers	51%	64%	45%	50%
Males (N)	352	66	124	162
% Smokers	37%	29%	44%	35%
% Non-Smokers	63%	71%	56%	65%

Table 3

Relationship Between the Smoking Behavior of College Students and the Smoking Habits of Their Older Siblings

	Students With Older Siblings	Neither Brother Nor Sister Smoked	Brother Or Sister Smoked	Brother And Sister Smoked
Females (N)	398	157	186	55
% Smokers	48%	29%	59%	71%
% Non-Smokers	52%	71%	41%	29%
Males (N)	223	94	109	20
% Smokers	39%	28%	47%	55%
% Non-Smokers	61%	72%	53%	45%

Table 4

Relationship Between the Smoking Habits of Parents and Older Siblings and the Smoking Behavior of College Students

	No Older Sibling Smoked[1]			Older Brother and/or Sister Smoked		
	Neither Parent Smoked	One Parent Smoked	Both Parents Smoked	Neither Parent Smoked	One Parent Smoked	Both Parents Smoked
Females (N)	79	124	160	40	94	106
% Smokers	22%	47%	46%	65%	65%	57%
% Non-Smokers	78%	53%	54%	35%	35%	43%
Males (N)	46	75	103	21	48	59
% Smokers	13%	37%	33%	62%	54%	39%
% Non-Smokers	87%	63%	67%	38%	46%	61%

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[1] Students with no older siblings and those whose older siblings were non-smokers are included in this category.

Table 5

Relationship between Parental Smoking Habits and the Maintenance of Students' Smoking Behavior

	Total Number Of Subjects	Neither Parent Smoked	Mother Or Father Smoked	Both Parents Smoked
Females (N)	295	43	119	133
% Current Smokers	40%	53%	34%	40%
% Occasional Smokers	26%	26%	28%	25%
% Ex-Smokers	34%	21%	38%	35%
Males (N)	130	19	54	57
% Current Smokers	31%	11%	26%	42%
% Occasional Smokers	29%	42%	35%	19%
% Ex-Smokers	40%	47%	39%	39%

Table 6

Relationship Between the Smoking Habits of Older Siblings and the Maintenance of Students' Smoking Behavior

	Total Number Of Subjects	Neither Brother Nor Sister Smoked	Brother Or Sister Smoked	Brother And Sister Smoked
Females (N)	193	46	108	39
% Current Smokers	43%	37%	48%	36%
% Occasional Smoking	27%	33%	24%	28%
% Ex-Smokers	30%	30%	28%	36%
Males (N)	88	26	51	11
% Current Smokers	30%	39%	18%	64%
% Occasional Smokers	35%	42%	37%	9%
% Ex-Smokers	35%	19%	45%	27%

International Review of Applied Psychology, in press.

On the Relation Between Family Smoking Habits and the Smoking Behavior of College Students

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